

Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING

THE INDEX OF OTOLARYNGOLOGY

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VOL. XL.

MARCH, 1931.

No. 1.

I.

IMMUNITY IN OTOLARYNGOLOGY.*

RALPH A. FENTON, M. D.,†

PORTLAND, OREGON.

Segregation of medical teaching and research into "laboratory" and "clinical" departments has brought about a segregation of literature, and within the literature a barrier of special terminology and definitions which makes it almost impossible for the busy practitioner to delve into recent publications in hematology, immunology and allergy without feeling lost and a bit scared. Lord Moynihan recently remarked, "Physiologic and other forms of research are drifting further and further away from clinical medicine, to the real disadvantage of both."¹

The otolaryngologist faces the difficult clinical job of diagnosis and treatment in a region of extraordinary complexity. "Confronted by a condition and not a theory," he must produce results; he must distinguish between endocrine imbalance, psychic

*Read before the Western Section Meeting, American Laryngological Rhinological and Otolological Society, Seattle, Wash., January 31, 1931.

†From the Department of Otolaryngology, University of Oregon Medical School, Portland, Oregon.

disturbance and local manifestations of infection or of neoplastic growth. People are sick and must be made well; and the physician who keeps abreast of clinical advances has little time or patience to familiarize himself with the new vocabulary (often varying with the laboratory in question), necessitated by advances in the theory and practice of immunology and bacteriology.

Yet we must be availed of these advances; for, as Oliver Wendell Holmes told the Massachusetts Medical Society, in 1860, "Disease, being always an effect, is always in exact proportion to the sum of its causes,"² a very pertinent reminder, not only that foci of infection may exist elsewhere than in the head, but also that every cause must be considered in making up the diagnosis.

May I take the liberty of talking over with you some of my own puzzles regarding immunity? One wonders why one child falls sick and its brother and bedfellow does not; why cancer selects the larynx of this smoker and not that; why the fulminant jugular thrombosis for one red throat, and only a pink drum membrane for another in the same epidemic.

Along with other students from all over Chicago, I listened awe-struck in 1905 to Professor Ehrlich, understanding not too much of his meticulous German demonstration of the side chain theory. Here was the new demigod of science, whose intricate theory of immunity, built upon the interlocking of receptors, complement fixation and all the rest, was to revolutionize medical science. Now, after twenty-five years, hear this: "The Ehrlich side chain theory has never demonstrated that any organ or tissue is responsible for formation of any given antibody";³ and this: "I am convinced there is hardly an element of truth in most of the hypotheses of the Ehrlich theory."⁴

The specificity of germs in the etiology of accessory sinus disease has been vastly overstated; for we now know that a dozen different microbes may show up in culture, with almost identical gross and microscopic changes in the sinus wall. We know also that the sinus wall may be edematous today under lipiodol, and may look normal tomorrow when operated on; yet a pure culture of this or that pathogenic germ may all the time be present. Such findings certainly do not fulfill the postulates of Koch.

One constant set of factors remains for consideration, if we lay aside for such reasons the specific germ theory and its corollary, the specific antigen theory, namely, the living cells of our bodies. Cellular physiology and pathology (and this includes the circulating tissues, blood and lymph) have received more special study of late than almost any other field in medical research. Unfortunately for us, this has meant the creation of a mass of new and special processes, nomenclatures and descriptions, mainly in the hands of technical specialists remote from the care of the sick. Fortunately, some of these gifted research men are willing to translate and abstract their findings in terms we can understand and apply to our problems. Wright of London, Kolmer of Philadelphia, Gay of New York, the late Maximow of Chicago, Zinsser of Boston, Benson and Sears of Portland, and others, have been most helpful in this regard. Our own philosophers, the lamented Ross Skillern, Mosher, Lewis Coffin, Levy, Emerson, Coates, Beck, Barnhill, Eugene Lewis—have looked beyond the immediate needs of surgical technic and antiseptic treatment to anticipate and to inspire important work in the field of immunity.

We inherit from our parents our natural immunity to disease, but with the first breath, the first food, we are exposed to outside influences which call into play the complex mechanism of acquired immunity. In response to external stimuli or irritants, what Zinsser calls an emergency mechanism specifically changes the reaction capacity of our tissue cells. In response to bacteria and their toxins, so-called antibodies are elaborated and begin to circulate in the blood. Yet in allergic conditions, where the specific change in cell reaction—hay fever, asthma, urticaria—is certainly very profound, no determinable circulating antibodies can be detected.⁵

According to Gay, "natural or innate immunity may in general be ascribed to the activity of the polymorphonuclear leucocytes of the circulating blood. Acquired immunity . . . involves certain new and enhanced physiological activities . . . expressed in the reaction products of serum, usually designated 'antibodies.'" But, he continues, "smallpox and typhoid immunity are neither due to polymorphonuclear leucocytic activity nor the action of antibodies. By exclusion, we are forced to con-

sider the fixed tissues of the body to find an explanation for certain phases of immunity."

Careful perusal of Connor's splendid monograph on laboratory aids in otolaryngology⁶ and of Shea's fine paper on blood changes in the anginas⁷ will prove very useful to all of us. The source of phagocytes and of the various types of leucocytes has been variously located; we have so far only to explain and to forecast changes in immunity by careful and repeated differential white counts. Much emphasis is rightly laid on the "shift to the left," as disclosed in such hemograms, toward "immature" leucocytic forms as leucopenia increases.

A curious reversal in the laws of immunity holds sway over allergic persons. Such people are abnormally active in the production of antibodies; not only do they provide their own antitoxins to germs, but also antibodies for pollens, foods, dusts, danders and a host of other substances. Such excessive antibody production results in sensitization of certain body cells, a process favored by the unstable vasomotor balance found in these individuals; and the sensitization of these cells is actually responsible for the disease (Kolmer).⁸

Fungi, such as the molds, thrush, trichophytosis, actinomycosis and blastomycosis are of relatively superficial growth and of doubtful specificity as to the creation of antibodies and of immune reactions. Their detection in our field by serologic and even by microscopic methods is often very difficult. Clinically, however, we are aware how readily skin and mucous surfaces may be softened and made vulnerable to subsequent bacterial invasion by these usually undiagnosed advance agents of trouble.^{9 10 11 12}

Fixed mesenchymal cells with marked phagocytic properties were long ago disclosed by Aschoff-Kiyono,¹³ in studies of the spleen, liver and bone marrow. When vital stains like trypan blue or finely divided masses like india ink are injected into the blood stream or into body cavities, such cells, which are modified forms of connective tissue, pick up the stain or the mass as part of their phagocytic task. By this so-called "blocking" of such cells, they may be readily detected wherever located in the body of the experimental animal. When saturation of such cells by the blocking agent has occurred, no further phagocytosis is possible for

varying lengths of time. For example, massive intravenous india ink injection in guinea pigs and rabbits prevents subsequent injections of diphtheria antitoxin from appearing in the blood for three weeks and more.¹⁴ In sensitized animals there is a drop in the quantity of complement in the blood fifteen minutes after ink injection, which reaches its low point after three hours and does not return to normal for twenty-four hours.¹⁵

Massive accumulation of reticuloendothelial cells such as these may be caused within serous cavities by injection of heavy sterile oils, aleuronat and the like. Such thickening of the pleura and peritoneum, experimentally produced, has rendered them impervious to a hundred times the lethal dose of hemolytic streptococci, quickly fatal in control animals.¹⁶

The reticuloendothelial system is widely distributed throughout the body, especially in perivascular connective tissue, where it is in close relationship with the vasodilator and vasoconstrictor nerve endings. The whole process of repair after infection or injury, with its influx of histiocytes, or small round cells, calls these structures into action. Dr. Larsell and I are investigating what proportion of such cells may be found in the normal, inflamed and infected sinus mucosa of experimental animals, and we hope to present some findings at a later date. Italian observers have done much work in this field, and have noticed that functionally active structures, especially where directly exposed to infection or trauma, seem to take up more of the stain than do interstitial, supporting or functionally inactive parts of the nose and throat.^{17 18}

There is practically constant desquamation of endothelial cells (clasmatoocytes) into the circulating blood of the rabbit and of man. These are not monocytes, but range from the smallest endothelial cell up to big stellate cells of the Kupffer type.¹⁹ In areas of irritation and congestion it is also possible to observe pale undifferentiated perivascular connective tissue cells migrating to and through the endothelial lining.²⁰

The omentum, which is known to attach itself to injured intraperitoneal structures is especially rich in reticuloendothelial cells. Charlton of Pasadena²² has secured an extract from such cells in the omentum of young pigs, with which remarkable stimulation

of the general defense mechanism seems to occur, suggesting somewhat the effects ascribed to the so-called "cancer serum" of Coffey and Humber. These factors in specific cellular defense are directed in normal persons against all sorts of extraneous dangers, irritants and toxins. Their presence in the skin explains the immunity of that organ to pure cultures of the most virulent organisms externally applied.

Recent work of d'Herelle on bacteriophage and of Besredka on mass culture vaccines, utilizing products of bacterial autolysis secured by reinoculation of the same species upon filtrates of previous cultures, is thought by Hays, Novak²³ and others to promise more in the way of stimulating local immunity by local use of the bacteriophage or of the mass vaccine than has been secured in the past by hypodermatic injection of autogenous vaccines or of alien proteins. Unfortunately, most of the success with these preparations has come in the treatment of dysenteric or pyodermic disorders, and not in the management of streptococci or other hemolytic organisms which invade our field.

Time does not permit a discussion of biochemical and physical factors in otorhinologic immunity, which include not only unfavorable environment,²⁰ but especially endocrine dysfunction,²⁴ vitamin imbalance,^{25 26 27 28} and the metabolic upsets incident to heart, kidney and gastrointestinal disease.²⁹ Barnhill, Dean, Shurly, Stucky, Daland and others have recently made important contributions to this phase of the subject.

Valuable adaptations of physical and electrochemical methods of influencing cellular growth and repair may develop from work on the regeneration of mucous membranes which is now in progress in several laboratories. The difficulties of clinical utilization of such agents in our field are numerous and obvious. Promotion of cellular resistance by diathermy, by zinc ionization, by mercury vapor lamps and quartz lamps, are familiar instances.

We are prone to forget that the exposed airways of the nose and throat are endowed with a higher immunity than the sheltered mucosa of the mastoid and the accessory sinuses. Shambaugh once humorously suggested that in cases of focal infection we might almost as well remove the septal or turbinal mucosa as strip out that of the antrum, both being equally infected. But

sinus mucosa has only the feeble resisting power of all disappearing structures, whose function is no longer necessary to the human economy. If needs be, let it be surgically removed, for within the new granulations and the new lining will be elaborated a tremendous volume of immunizing substances. Over such raw surfaces, following Mosher and McGregor, recent observers have repeatedly established the fact of regrowth of ciliated epithelium, properly provided with glands. However unphysiologic it may seem at first, good surgery is thus an immunizing process.

Nevertheless, should it prove possible to secure regeneration and reactivation of sinus and ear mucosa by improving cellular chemistry; by controlling the circulation through the sympathetic system; by stirring up the reticuloendothelial cells to an improved defense: we may have less and less radical surgery to do. Restoration of physiologic function and a distinct limitation of loss of tissue will probably be corollaries of such future conservative surgery, which will be based upon more accurate conceptions of immunity than any now available. To quote Lord Moynihan: " . . . It is in the application of the methods of both Hippocrates and Galen, in observation, critical judgment and discrimination, followed by hominal and animal experiment, that we shall most surely attain to greater and even greater knowledge of the maladies of men, and so acquire power to conquer or subdue them."¹

MEDICAL ARTS BUILDING.

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II.

HAS SCIENCE THROWN ANY NEW LIGHT ON OUR UNDERSTANDING OF CHRONIC PRO- GRESSIVE DEAFNESS?

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In previous articles the writer tried to show that when a focal process caused a tubotympanic infection accompanied by a loss in tone perception, there were two things that happened.

First—In one there were tissue changes in the tube and middle ear which interfered with the transmission of sound waves through the conduction apparatus. In the other, there was loss of tone perception due to a bacteremia affecting the end organ through the blood stream which was shown by tinnitus which always recurred with any exacerbation of the local process, such process, in the experience of the writer, almost invariably was due to a closed follicle in a chronically infected tonsil.

Second—The progress of the disease was intermittent and always due to the lighting up of such focal infection, as indicated by the sensitiveness of the sentinel gland to palpation on the corresponding side. The exacerbations, which were followed by the greater loss in tone perception, were due to congestion, increased secretion and tension within the follicle followed by discharge into the blood stream.

This might go on indefinitely or after such a discharge the drainage might so far improve that the chronic infection would result in fibrosis and round cell infiltration, but was of a mild type without noticeable exacerbations, and the progress of the deafness was very slow.

Third—The course of the disease in chronic cases which was not disturbed by a virulent intercurrent infection is the same for all types involving the middle ear, including primary nerve deafness which showed no middle ear pathology. If a virulent infection should take place after the acute symptoms have subsided the further course is the same as in all chronic cases.

Fourth—A negative Rinne occurs when the loss of tone perception has advanced to the same degree in all forms of middle ear disease as well as in nerve deafness.

Fifth—The end result in all cases of chronic progressive deafness is nerve deafness if the disease advances far enough. The study of middle ear disease accompanied by deafness has been based in the past on the atomic theory, in which molecular waves were transmitted through the conduction to the perception apparatus where they were recognized as sound. Where there was loss in tone perception it was thought to be due to gross pathology in the middle ear which interfered with the movement of the ossicles in their transmission of sound waves to the organ of Corti. Notwithstanding the fact that tone perception is always impaired throughout the scale, as shown by the audiometer, although the higher notes are not influenced by middle ear obstruction. In addition to this fact, no nerve injury was supposed to be indicated by the marked tinnitus that was present in all cases.

We must admit that this conception of chronic progressive deafness has not resulted in the cure of a single case in the past fifty years. With this background, it should be permissible to advance some other theory in explaining the loss of tone perception which the writer has attempted to do in previous papers whose summary is given at the beginning of this article.

Now, has science, which has made such rapid advance in the past twenty-five years, contributed anything that could be taken to support one theory or the other?

Scientists, including astronomers, physicists, mathematicians and chemists, all agree that the atom is further divisible into many electrons and that electronic vibrations produce sound waves. Where these vibrations are slow they are recognized as motion, the next increase in rapidity of the vibrations gives us the sound area followed by red light, and then all the colors of the solar spectrum. Above this we have electricity, ether and cosmic fire.

The astronomers have proved that the seething mass of electrons that form the center of the sun have polarity, showing attraction and repulsion.

The final conclusion of many scientists is that the difference between organic and inorganic substances is simply one or two more electrons of carbon. (Sir James Jeans.) So that sound under all circumstances is the result of electronic vibrations from whatever cause. The sound receptors are numerous, but all nerve impulses are propagated by electricity. (Reymond.)

We at once see the bearing of such dynamic vibrations in our study of slowly progressive deafness, for a given nerve always propagates the same kind of impulse, irrespective of its stimulus. (Crile.)

It is obvious that we cannot interpret nerve dysfunction in terms of middle ear pathology, for physical obstruction is constant, while the loss of tone perception varies from time to time and often clears up for a year or more after the removal of the focal infection and is finally lost following an acute exacerbation of the same process. To account for the phenomena which characterizes slowly progressive deafness we must have some factor that is active throughout the life of the individual and capable of causing recurring trauma or infection of the acoustic apparatus.

Furthermore, as nerve deafness is apparent in all cases when perception has been lowered sufficiently and is the end result, we must assume that perception was being impaired from the beginning coincident with the organization of the gross tissue changes in the middle ear.

The passage of electronic waves through the ear activating the organ of Corti would produce sound. Is it not a fair assumption that tinnitus, which ushers in the loss of tone perception and cries out to us throughout its course, is due to irritation of such atomic nuclei?

We know that an electric current passes from one pole to another without regard to the intervening obstruction, so that any loss of tone perception is more apt to be due to the gradual damage to the nuclei than to any obstruction in the middle ear.

The electrons forming such nuclei of all cells, whether in the brain, nerve or muscle, gives us an idea of where the damage occurred in the transmission of impulses from the periphery to the brain in our study of slowly progressive deafness.

Scientists are doing some very valuable work in restoring the function of these electrons which have been impaired by infection or trauma, especially in plants, which should be helpful to us in the treatment of deafness after removing the cause.

CONCLUSION.

To answer the question embodied in the caption of this article, Science has strengthened the contention that slowly progressive deafness is a disease involving the perception apparatus. The concomitant changes in the tube and middle ear have, in the opinion of the writer, no relation to the nerve dysfunction. The recognition of electronic nuclei does not disturb our statement in regard to the etiology and progress of the deafness, but does show clearly that our former ideas of tinnitus will have to be changed. Science has also shown very definitely how tone impulses are propagated.

III.

THE USE OF SURGICAL DIATHERMY ABOUT THE MOUTH OF THE EUSTACHIAN TUBE FOR INFECTION AND CATARRH OF THE MIDDLE EAR.*

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The pharyngeal orifice of the eustachian tube is funnel shaped and bounded above and behind by a prominent ridge called the eustachian cushion. There is a slight ridge which descends from the inferior end of the tube to the side wall of the pharynx. The levator veli palatine muscle in descending runs parallel with the tube and along its lower border.

The pharyngeal recess, or fossa of Rosenmüller, is an almost vertical depression which extends laterally in the form of a flattened pouch. It was once considered to be the remains of the pharyngeal portion of the second visceral cleft, but it is formed later, and therefore does not arise from the second cleft. The pharyngeal recess projects laterally above the superior margin of the superior constrictor muscle, and below the petrous portion of the temporal bone, sometimes to the depth of three-quarters of an inch. In the recess is found a considerable amount of lymphoid tissue.

In hypertrophy of the adenoid, the pharyngeal recess (fossa of Rosenmüller) usually contains much hypertrophied lymphoid tissue, and even after an adenoidectomy has been performed there may remain much infected lymphoid tissue in the recess. Very frequently, in postoperative adenoidectomies, where trauma has taken place in the endeavor to clear out this space, there are a number of adhesions from the eustachian cushion to the post-pharyngeal wall, which close in the infected lymphoid tissue behind them in the pharyngeal recess.

*Presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Atlantic City, January 5, 1931.

Also, there may be many lymphoid follicles on the anterior and posterior lips of the eustachian tubes, and even within the mouth of the eustachian tube itself.

There is a ridge of mucous membrane descending from the inferior end of the eustachian tube on the side wall of the pharynx that is fused with the lateral pharyngeal fold and which may be thickly studded with lymph follicles.

This infected lymph tissue in the pharyngeal recess, and on the tubal lips and below, extending into the lateral fold, frequently is the cause of changes in the middle ear, varying from a mild tubal catarrh to an acute purulent otitis media, or especially, recurring acute purulent otitis media and chronic purulent otitis media.

The condition of infection in the lymph tissue about the tube has long been recognized, and the oldest method used to correct the condition was to break down the adhesions with the finger, assuming that the infected lymph tissue would take care of itself: later, by the use of the Holmes or Yankauer nasopharyngoscope, the method was to pick or bite out the lymph tissue with forceps. This method usually tended to increase the adhesions and hence to make the condition worse rather than better.

The newer method which I wish to present to you is that of obliterating the lymphoid tissue in the pharyngeal recess and destroying the infected lymph follicles by electrocoagulation. This is done with variously shaped, insulated, active electrodes, with pointed or dull tips, used through a Yankauer nasopharyngoscope.

The technic of using the high frequency coagulating currents is hard to explain in definite terms. The amount of current necessary depends on the distance it passes through the tissues. That is, if a metal plate, as an indifferent electrode, is placed on the back or arm of the patient, it requires much more current than if the two terminals are close together. I favor having the terminals close together and using less current. The aim is to get fairly slow coagulation about the needle, taking about two seconds to obtain the desired amount of coagulation at each puncture. A white spot, four to five millimeters around the needle, and about one millimeter beyond the point, is thus made.

This procedure is carried out to all lymph follicles, if they are isolated, as they are on the tubal cushion. The needle is plunged into the center of the follicle and the current turned on until the follicle turns white. In the pharyngeal recess the first difficulty is that unless the nasopharyngoscope is passed very gently the adhesions will be torn and blood will interfere with vision as well as with coagulation. The adhesions should be destroyed before they are torn apart by the nasopharyngoscope. All lymphoid tissue in the pharyngeal recess should then be destroyed.

Coagulation of the lymphoid tissue below the tube must be done especially carefully or the result may be a paralysis of the levator veli palatine muscle. If the lateral pharyngeal fold contains much hypertrophied lymphoid tissue this should also be coagulated.

It takes from ten to fourteen days for the slough to disappear and about four weeks before complete healing is effected. The slough remains for nearly two weeks between the adjacent surfaces, and the burned surface, even after the slough comes away, tends to prevent recurrence of adhesions in the pharyngeal recess. While there is a possibility of starting up an acute ear from the sloughing surface at the mouth of the tube, this has not as yet occurred.

Considerable practice is required to gain a knowledge of how deep to insert the needle and what amount of coagulation should be done.

ILLUSTRATIVE CASES.

J. G., male, aet. 11. This is a typical untreated case, with a history of recurring earache. When first seen he had acute purulent otitis media with a temperature of 104, and the mastoid was tender. The ear was incised and his temperature dropped to normal in twelve hours. Fourteen days later the ear was dry. The patient had slight adenoid remnants and his parents were strongly urged to have the pharyngeal recess cleaned out. Instead the boy was taken back to the man who had previously performed the tonsil and adenoidectomy. He removed the adenoid remnants but evidently paid no attention to the pharyngeal recess, which had many adhesive bands from cushion to postpharyngeal wall. Examination last April showed the right attic bulging and

inflamed, but it gradually subsided. Both drums are extremely retracted and dull. The pharyngeal recess is filled with lymphoid tissue and adhesions. Audiogram shows loss, right 13.5 per cent. and left 13.5 per cent. The parents refused to have condition corrected.

C. M., male, aet. 7 years. Left drum dull; hearing down in the left ear about one-third; tonsil stumps and adhesions in the pharyngeal recess. Adenoid remnants were attached to the cushions with a small passage behind the cushions into the pharyngeal recess, all of which were coagulated.

M. C., female, aet. 9 years. Patient had a mastoidectomy four years ago. With each cold, which occur frequently, the right ear discharges pus through a perforation in the posterior inferior quadrant. Drum infiltration covers up all landmarks. The left ear shows a dry perforation. There is much lymphoid tissue and adhesions in the pharyngeal recess, especially on the right side. Large flat tonsils. Treatment: Removal of tonsils and small remnant of adenoid tissue; coagulation of the lymphoid tissue in the pharyngeal recess and lateral pharyngeal folds. Result: Right ear slowly dried up. Patient has had two epidemic colds since but the ear has remained dry.

A. C., male, aet. $8\frac{1}{2}$ years. Slight mental retardment. Tonsil and adenoidectomy four years ago, nasal obstruction (swollen inferior turbinate), frequent colds, dull drum. Diathermy to tonsil stumps, lateral folds, tubal and pharyngeal recess lymphoid tissue. Six weeks later patient was doing better school work, nasal breathing, appetite better, and a marked general improvement.

B. C., female, aet. 6 years. Three years ago patient had scarlet fever with double acute purulent otitis media. Tonsil and adenoidectomy three months later. Double mastoidectomy one year after the scarlet fever. One year later right secondary mastoid. Two years later secondary adenoidectomy. Six months later tertiary right mastoid. Since then intermittent discharge from right ear, with colds. Left ear, intermittent discharge, dry for the past ten months. General health said to be good. Hearing loss, right 41 per cent, left 38 per cent. Both drums completely destroyed. Nose negative. Hypertrophied lateral pharyngeal folds. Large

amount of lymphoid tissue in the pharyngeal recess. Tubal cushion very much enlarged. On the right side there was a large band of tissue from the anterior lip of the tube across to the near midline of the posterior pharyngeal wall with free space behind it. This was divided and reduced down to shape the anterior lip. It was impossible to find the mouth of the tube. All the lymphoid tissue was destroyed by coagulation, and four weeks later the ear was dry.

T. B., male, aet. 9 years. Several years ago the patient had a tonsil and adenoidectomy. When first seen he had acute purulent otitis media which had existed for ten days. Both ears ruptured and were incised three days later. He had a positive blood culture, streptococcus hemolyticus, four days later, and was having a chill followed by a high temperature each day. When first seen by me, which was on the tenth day, there was a slight discharge from the left ear. The right was dry and there were no mastoid symptoms. Temperature normal in the morning, and went to 104 in the afternoon. With a positive blood and history of double ears, it was impossible to determine which sigmoid sinus was infected. After running a septic temperature for ten days there appeared an induration of the lymph glands below the tip of the right mastoid. Mastoidectomy with jugular ligation was then performed and the sinus opened. Pus was found in the mastoid cells, no clot in the descending sinus. After operation the temperature remained normal and recovery was uneventful. Eight months later the patient had a coryza with pain in the left ear for one day. Spontaneous rupture of the drum membrane, with discharge for two days. On the thirteenth day the right ear became swollen and tender; temperature 102; drum membrane red and bulging. There was pain and tenderness in the right mastoid scar, which was red and bulging. The following day the old mastoid incision was opened, and half a dram of pus found in the antrum. Both drum membranes were incised. There were large masses of lymphoid tissue in the pharyngeal recesses and the nasopharyngeal vault; the tubal cushions were much enlarged. All lymphoid tissue coagulated. Ten days later the ears were dry, and in four weeks the mastoid wound was healed. Patient has remained well to date, a period of two years.

Adult. For the past four years patient has had attacks of "pressure" in left ear. Tonsil operation five years ago. Examination showed congested long recess, left ear. Many adhesions in the left pharyngeal recess were destroyed with coagulation. Three days later drum normal, tube open, no sensation of pressure. No trouble since (two years).

W. A., male, aet. 4. In 1922 patient had acute cervical adenitis. Tonsil and adenoidectomy one year before. In 1929 he had recurring acute purulent otitis media; discharging at present. Much lymphoid tissue on the posterior pharyngeal wall, lateral folds, pharyngeal recess and nasopharynx. Coagulation to tonsil fossæ, lateral folds, pharyngeal recess. Guillotine to large adenoid. The velum was immobile from burns around the levator palati muscle; this cleared up in three weeks. One year later the ear was dry and the drum very thin and retracted.

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IV.

THE SIGNIFICANCE OF HYDROPS MUCOSÆ.*

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LOS ANGELES.

To suggest anything new in the way of pathology of the upper respiratory tract should give pause to even the most intrepid. This year Dr. D. C. Jarvis of Barre, Vermont, published "The Upper Respiratory Tract as a Guide to Nutritional Disasters," a work representing very acute and painstaking observations of a great variety of upper respiratory conditions over a period of fourteen years. The data and deductions contained in Jarvis' paper constitute exceptionally constructive and practical contributions, the value of which will be increasingly apparent with the passage of time. From the standpoint of the patient's well-being, this may come to outweigh the contributions to radical rhinologic surgery during the same period. Fortunately, from other sources during the past two decades, material of similar import has been finding its way into medical literature and thought. The names of Vedder, Evvard, McCollum, Osborne, Goldberger, Hess and Gamble are associated with much of this work; it remains to analyze these various contributions and to apply the principles to various fields of specialized medicine. Among other rhinologists Shurly, Dean, Jarvis, Proetz and the writer have made some beginnings toward this end. This communication aims to call attention to what the writer has come to regard as a common pathologic entity of considerable importance in rhinologic practice, namely, *hydrops mucosæ*.

The data underlying the observations incorporated in this paper originated in the "mine-run" of patients presenting at four large clinics per week during the past nine months, a total of 1500 individuals of all ages and nationalities. A large number of these individuals had submitted to various upper respiratory surgical

*Read before the Western Section, American Laryngological, Rhinological and Otological Society, Seattle, Wash., January 31, 1931.

procedures including tonsillectomy, adenectomy, turbinectomy, various fenestrations and curettings of the antrum, bulla, frontal, ethmoid and sphenoid sinuses, and various degrees of removal of the lateral nasal wall structures. Many of them experienced postoperative alteration of symptoms for a longer or shorter period of time; but, without exception, they again sought relief from upper respiratory troubles.

It was on the occasions of their postoperative reappearances at the nose and throat clinics that the writer encountered them. Systemic treatment was devised and followed carefully in each case. Occasionally local pathology demanded some additional local treatment, such as removal of grossly obstructive polyp, tamponade or incision of acutely inflamed drumhead, nasal tamponade or hot applications; but, in general, systemic measures have been the only treatment applied. The local and general improvements noted in the cases have been striking; turgid swelling or edemas have subsided; pains have subsided; discharge has changed from copious purulent to negligible nonpurulent; improvement locally has been accompanied by other improvements, in weight, appetite, sleep, strength and general wellbeing. Unfortunately, there is no specific treatment or one set of prescriptions which may be presented. There are, however, certain fundamental concepts and working hypotheses which are sufficiently definite to present in connection with an outline of the physical characteristics of the constitutional condition under discussion. It seems futile to undertake any such systemic direction of a case until there has crystallized a definite working conception of the conditions which underlie tissue reactions, both in health and disease. Once crystallized, this working conception must serve to suggest the details of systemic treatment and check it by the clinical progress of each case.

For the past decade the writer's attention has been increasingly focused upon the changes in the upper respiratory mucosa which characterize (specific) "allergic" and (nonspecific) "atopic" tissue reactions. The outstanding change is the increased fluid content of the mucosa, regardless of the presence or absence of free secretion upon the nasal surfaces; it is not unusual to note

conspicuous absence of secretion upon a definitely edematous mucosa. The difference in texture of the mucosa is characteristic; it is pebbled, at times finely, at times coarsely, and pits very readily on pressure; it shows subnormal degrees of shrinkage to light touching with 1 per cent cocain, but never approaches normal full shrinkage to a close contact with underlying bone. The difference in color is notable; the usual redness pales toward "skimmed milk" or "blue-gray." The blue of the sky, the bluishness of skimmed milk, and the blue of bodies of water is attributed to the "Tyndall effect"; the peculiar difference in color of the mucosa characteristic of the condition under discussion is explained by the same physical effect.

The symptoms and sensations of patients in whom this condition exists are those, in general, of upper respiratory irritations, namely, sneezing, obstructed nasal respiration (often alternately in right and left nostrils), impaired sense of smell, feeling of fullness, even to the degree of headache, aprosexia, discharges varying from watery to viscid and from clear to grayish, yellowish, greenish or brownish. Coincidentally there frequently exist conjunctival, palatal, external auditory canal and skin areas of irritation, varying from itching and burning to more painful sensations; excessive tears and cerumen are very frequent, as are sodden, cracked epithelial areas about the canthi, the nares, the auditory canals and the mouth. That the underlying biochemical disturbance is not thus limited in its physical manifestations is attested by associated spastic colon, spastic ureter, mucous colitis, gastric ulcer, arthritis, hemicrania, urticarial and eczematous skin lesions and more extensive local edemas, such as of the feet and hands. Often the fundamental picture is obscured by secondary infectious incidents, with incidental toxicosis, fever and local tissue destruction. White blood cell count may show from 3 to 6 per cent or even more eosinophilia with no other abnormal findings, except those attributable to the infectious secondary incidents. The writer has records of a large number of such cases in whom extensive skin tests have failed to show allergic reactions, yet the clinical course and physical examination reveal unmistakable definite special tissue sensitivity and hydrops mucosæ.

One of the special characteristics of such patients is their facile change in weight—often as much as two or three pounds in a single day. This fluctuation is due to altered amounts of fluid in the body tissues. This is further borne out by finding at times otherwise unexplainable discrepancies between the amounts of fluid intake and output. There seems to be a noticeable mental alertness and keenness characteristic of a large number of these patients. Aprosexia, with obtunded mental alertness and memory, is noted during or after acute inflammations, especially those with secondary infections. Occasionally in adolescents, rarely in children, frequently in adults, one encounters various degrees of polyp formations in the nasal and sinus mucosa; the polyps are usually multiple, pellucid and small. They differ in texture and size from the fibrotic organized polyps occurring at sites of chronic mucoperiosteal infectious lesions. The mucosal linings of nasal accessory sinuses, especially the antra, are frequently found, either by operative inspection or in the X-ray, to be polypoid. The writer has seen a gelatinous, translucent antral lining approximately one-half inch in thickness, and has watched this same mucosa subside to its former gossamer-silk texture and tight application to the antral periosteum within the period of a few days as the edema subsided.

History may succeed in eliciting a family tendency to something of the general nature of epithelial or glandular manifestations of disturbed function, ranging from eczema, hives or seborrhea, through arthritis, colitis, bronchitis, asthma, hay fever, conjunctivitis, hyperceruminosis, epilepsy, ureteral or biliary colic, to dysthyroidism or other endocrine imbalance. There are distinctly hereditary traces to be found in most cases, not necessarily duplicating the upper respiratory picture but relating to it through one or more of these conditions.

Local tissue changes manifest disturbed hygroscopy, of varying degree and extent, and of wide range in symptomatology. The only force capable of bringing about such local fluctuations in tissue fluids is osmotic pressure. Such profound variations of osmotic pressure within relatively short time intervals suggest electrolyte rather than colloid change.

No working conception of this condition is to be had without collateral considerations of hydrogen ion and electrolyte concentrations in the tissues, hormone balance fluctuations, varying vitaminosis and antigenesis; further correlations must take cognizance of a considerable category of ills representing family tendencies to unusual tissue reactions. In general, these show disturbances of epithelial and glandular tissues, not necessarily associated with clinical toxicosis. At times infection may be superimposed upon the basic departure from normality to the extent of altering the primary clinical picture beyond recognition. The initial biochemical wrong, nevertheless, remains as the *essential* etiologic factor and as the essential point of attack for rational therapy.

Hydrops mucosæ is one of the manifestations of a constitutional peculiarity. One finds in the German literature that Czerny has described the "exudative diathesis," others refer to "habitus exsudativa." The work of Duke-Elder, the studies of edema by Fischer and Alwyn, Kruyt's book on "Colloids," the Transactions of the "Colloid Symposium" and Zondek's "Electrolyte in Biology and Pathology" may be mentioned among recent sources of information bearing directly upon the physics, chemistry and biology concerned. The rôle played by lipoids in cellular morphology has been the subject of extensive investigation and speculation. Recently Crile demonstrated the organization of distilled water, salts, protein and fats into cell forms which undergo fission, manifest pseudopodiosis, consume oxygen, give off carbon dioxide and otherwise behave like living cells. His observations suggest that, under certain conditions, lipoids have the power to select and bring together in organic relation the constituents of animal cells. Wright showed that lipoids determine variations in surface tension of living cells. Variations in surface tension are concerned in the phenomena of permeation of tissues by bacteria, phagocytes and inert particles, such as coal, lime and iron dust. Redslob, Baumann, Fischer and Alles have shown that alterations in intraocular tension occur with changes in electrolyte content of the vitreous and aqueous.

Comprehensive discussion of these matters would be beyond the ability of the writer and outside the limits of this paper.

Physical and chemical researches are referred to thus briefly in order to touch upon some of the sources one must look to in seeking to understand the nature of the biologic changes under consideration.

The clinician in any field of medicine today finds himself confronting a mass of chemical and physical findings which bears directly upon his major clinical problems in the diagnosis and treatment of illnesses. The time has passed for a physician to disclaim knowledge of these data on the ground that they lie outside of the limits of his field of work. Knowledge of the biophysical and biochemical changes which constitute life phenomena must occupy first place in the mental equipment of the physician, no matter what his field. Physiologic and pathologic phenomena are generically identical in that they are manifestations of physical and chemical changes in living tissues.

"Facilis descensus averni" is nowhere more convincingly exemplified than in disease. The sequence of changes ensuing upon the initial departure from normality—be it overexertion, traumatism, exposure, dietary indiscretion or what not—reveals a series of ever-increasing physical and chemical disturbances of normal tissue activities; the disturbances affect metabolic, circulatory, nervous and endocrine balance, with resulting changes in hydrogen ion and electrolyte concentrations, free and bound fluids, surface tensions and osmotic pressures; hitherto effective barriers are lowered, permitting bacterial invasions.

(It is possible that the writer should have included among the just mentioned, "initial departures from normality," the item "infectious contacts." The South Sea Islander succumbs to simple contact with the infection of measles, the Eskimō to simple contact with the tubercle germ. Clinical experience seems to show that simple contact suffices to establish infection with gonococcus; perhaps there may be others, but, in general, this is not true of most of the pathogenic organisms commonly met with in acute infectious disease.)

The presence in the tissues of these living foreign bodies and the dissemination of their toxic products add tremendously to the physical and chemical disturbances which constitute the pathologic picture. Breaking down of tissues—both of the body and

of the invading organism—is an additional source of noxious material which further complicates the situation. The composite effect of all factors determines the outcome, responsibility for which is but rarely attributable to the initial departure from normal. Analysis of the mechanism of disease points unmistakably to the possible reversibility of the process at many stages, depending upon varying conditions in each individual case. Convalescence and recovery are biologic phenomena of reversal of biophysical and biochemical processes. The individual constitution is obviously of tremendous importance in determining the results which may follow an initial departure from normality. This paper deals with a common type of constitutional abnormality, one of the frequent manifestations of which is hydrops mucosæ. The etiologic significance of this underlying constitutional condition is not generally recognized, either in connection with upper respiratory troubles or with others of the same general nature.

Systemic therapeutics calculated to alter the peculiar constitutional tissue lability manifested as hydrops mucosæ is of first importance in solving the problems of many of the irritative, inflammatory and infectious conditions of the upper respiratory tract.

1154 ROOSEVELT BUILDING.

V.

THE USE OF OMNADIN IN OTOLARNGOLOGY.*

WM. MITHOEFER, M. D.,

CINCINNATI.

In 1911, Fraenkel and Much demonstrated the fact that the injection of the lipoid of bile had a beneficial action on experimental gall bladder infection of animals. Later on, Much and Schmidt experimented with protein of nonpathogenic bacteria and fat, and, with the addition of lipoid of bile, produced the preparation which they called Omnadin. Omnadin, therefore, contains the protein of nonpathogenic bacteria, lipoid of bile, and fat—three agents capable of increasing the nonspecific immunity of the body.

According to Much, the lipoid contained in this mixture is of greater value than the fat and protein. He is undoubtedly correct when he states that proper lipoid metabolism plays an important rôle in the establishment of immune bodies, and that it has a beneficial influence on the action of the endocrine glands.

The presence in an individual of a nonspecific immunity will be the means of preventing infections of the upper respiratory tract. The bacteria entering the body of such an individual meet with a resistance not present in one of the opposite type. It is of paramount importance, in treating any of the infections of the ear, nose and throat as well as those affecting other parts of the body, to increase, if possible, the nonspecific immunity of the patient in the early stage of the disease, at which time the immunity of the patient is at its lowest ebb. It is at this period of the disease that the injection of Omnadin is of great service, inasmuch as it produces a nonspecific immunity a few days before it is ordinarily established. This is the reason why we often see, within a few days after its use, a complete disappearance of what appeared to be a severe infection.

*Read before the Middle Section of the American Laryngological, Rhinological and Otological Society, Ann Arbor, January 17, 1931.

The intramuscular injection of Omnadin produces a mild reaction of the cellular elements of the body; in other words, a teasing dose instead of an overwhelming one, which often follows the injection of the various milk preparations. This fact makes this preparation most suitable for use in the acute stage of an infection associated with high fever. The mild, almost unnoticed reaction which follows its injection makes it a valuable agent in the treatment of infections occurring in childhood. In a recent case of severe erysipelas of the ear in an infant nine months old, the daily injection of one-half of an ampule (1 cc.) of Omnadin stopped the spread of the disease within two days and caused a disappearance of the fever at the end of the third day. Another point which speaks in favor of its use is the fact that anaphylactic symptoms are unknown.

In reviewing the literature, I was favorably impressed with the fact that good results following the use of Omnadin were also being reported in influenza, pneumonia, puerperal sepsis, encephalitis, cholecystitis, uveal tuberculosis, etc.

Autogenous and stock vaccines are said to be specific in their action. I doubt very much if this is the fact, insofar as I have seen furunculosis of the ear disappear in the same space of time after the injection of Omnadin as it did after the use of a staphylococcus stock vaccine. May we not assume, therefore, that the staphylococcus vaccine has no other effect on the cells of the body than the establishment of a nonspecific immunity? This is precisely the same action that is attributed to Omnadin.

During the past four years we have given over 5000 injections of Omnadin. The injection is given intramuscularly, either in the gluteal or deltoid muscles. Patients have never complained of a local or general reaction following its use. When given in the deltoid muscle, there is apt to be a slight pain for a few hours after the injection. Many patients have stated that they felt very much stimulated, and that their appetite for food had returned after having had Omnadin. We are in the habit of giving a daily injection of one ampule (2 cc.) for three successive days. Except on rare occasions is it necessary to give more than three or four injections. In severe infections we have not hesitated to inject the contents of one ampule, twice daily.

We have seen good results after the use of Omnadin in the following diseases:

Nasopharyngitis.—In the early stage of this disease the administration of Omnadin very often acts as a specific. Especially is this true when the disease is sudden in its onset and accompanied by severe pain on swallowing. When the nasopharyngitis is slow in its onset and is but a local expression of some systemic disturbance, such as intestinal toxemia, Omnadin has little effect unless other measures for the relief of the intestinal derangement are instituted. Nasopharyngitis is, as you know, many times accompanied by a leukopenia, especially during the first few days of the disease. The increase in the number of leucocytes five hours after the injection of Omnadin undoubtedly accounts for the good results obtained.

Peritonsillitis.—In the early stage of this disease we have often seen complete disappearance of the peritonsillar inflammation following the second or third injection. The same is true in acute tonsillitis. We have all undoubtedly made the observation that occasionally there occurs, about the fourth or fifth day following tonsillectomy, a painful inflammation of the soft palate, especially in the region of the posterior pillar. If the inflammatory reaction persists, there is danger of the formation of a retropharyngeal abscess and general sepsis. We have seen, on several occasions, a complete disappearance, within a few days after the injection of Omnadin, of a severe inflammatory reaction of the tonsillar wound, and would, therefore, recommend its use whenever the tonsillar wound is of abnormal appearance.

Vincent's angina, when accompanied by a thick, membranous deposit upon which salvarsan and chromic acid have little action, responds well after the use of Omnadin. A very unusual case of this kind was that of a boy, sixteen years of age, who had been bedfast for two weeks when I first saw him, suffering with a sore throat and an evening temperature of 101 to 102 degrees. Upon examination, the subtonsillar area on the right side was found to be covered with a grayish membrane which extended downward and involved the right half of the epiglottis. Culture was negative for diphtheria; the blood picture was not that of agranulocytosis, but the smear was positive for Vincent's angina.

It seemed futile to apply chromic acid because of the thickness of the membranous deposit. After the third injection of Omnadin there was no more fever, and a few days later the membrane had entirely disappeared. When healing was complete it was found that a part of the right half of the epiglottis was missing. We have never seen a more severe infection accompanying Vincent's angina.

Acute Otitis.—We use Omnadin as a routine in all of our acute infections of the middle ear and feel that by its use we have often cut short the progress of the disease. Many patients report relief of pain after the injection. In exudative catarrh of the middle ear it has also been found to be of benefit.

External otitis and furunculosis, and eczema, especially the acute form, respond well after the use of Omnadin.

We wish likewise to recommend Omnadin in all patients in whom a lowered resistance is suspected, especially in diabetic subjects, and in the fortunately rare cases of hemorrhagic mastoiditis where early operative interference is found necessary. It is important in such individuals to increase their nonspecific immunity before operation.

In the treatment of acute nasal accessory sinus disease, we have found Omnadin a valuable adjunct. In these cases we are in the habit of giving daily injections for at least a week or ten days, in conjunction with other forms of treatment. If there is present a very severe infection of the maxillary sinus three injections of Omnadin are given before proceeding with irrigation. There is less reaction following the lavage if this is done.

We have, furthermore, as a routine measure given a daily dose of Omnadin for the first three days following an operation on the nasal accessory sinuses and after mastoidectomy, and feel that by so doing we have increased the nonspecific immunity of the patient at a time when it is most needed.

Our experience with Omnadin has impressed upon us the fact that it is a valuable agent in the treatment of streptococcic infections. Any agent which stimulates the cellular elements of the body so that the constitution of the individual is placed on the defensive is worthy of consideration.

19 GARFIELD PLACE.

VI.

PULMONARY COMPLICATIONS ASSOCIATED WITH THROMBOSIS OF THE SIGMOID SINUS: REPORT OF A CASE.*

HAROLD I. LILLIE, M. D.,

ROCHESTER, MINN.

Remote metastatic manifestations of septicopyemia secondary to suppurative disease of the mastoid are usually of grave significance. It is sometimes difficult to determine whether the remote manifestations are secondary to the disease of the mastoid or whether they represent another manifestation of the original general infection, especially if the occurrence is noted early in the course of the disease. If the secondary focus occurs some time after the onset of the primary infection, it can be inferred usually that it was blood-borne. Direct extension of the original infection usually is caused by continuity along the respiratory tract and occurs early in the disease.

Fortunately, pulmonary complications in operable cases are relatively rare. In a series of 933 cases observed at the clinic in which operation was performed on the mastoid, disease of the sigmoid sinus occurred in eighty-eight. In four of these eighty-eight cases a pulmonary complication developed immediately before or after operation. In one case, a pulmonary infarct developed in a case in which the sigmoid sinus was injured at operation. In one case pleurisy with effusion was known to exist before operation; as surgical interference was urgent, the operation was performed under local anesthesia. In one case pleurisy with effusion developed after operation. The fourth case is reported in this paper. It is reported because of its interesting features and because a definite diagnosis was not made. The patient recovered.

In the case in which an infarct occurred suddenly on the seventh postoperative day there was complete recovery. At no time

*Read before the Midwestern Section of the Laryngological, Rhinological and Otolological Society, Omaha, Neb., January 31, 1931.

were there any signs or symptoms of sepsis, so it is probable that the thrombosis resulting from injury to the sigmoid sinus did not produce infection. The initial symptom was severe pain in the right lower side of the thorax posteriorly. Neither a rise in temperature nor a change in the blood picture occurred. Roentgenograms revealed what was thought to be extensive pneumonia. Pneumonia, without fever, increased leucocyte count or apparent illness did not seem plausible, and therefore a diagnosis was made of pulmonary infarct. The two patients who had pleurisy with effusion, one before operation and the other after operation, recovered although the convalescence was protracted. Nothing was done to the thorax.

In two cases a pulmonary complication was present which was not related to operation. One patient with extensive embolic pneumonia, mastoiditis and sinus thrombosis, late in the course of scarlet fever, was seen in extremis and therefore operation was not carried out. In the other case pneumonia and empyema developed after operation and the patient died. It is probable that there was no connection between the pneumonia and the operation, as the patient had been dismissed from the hospital two weeks previously and was being seen in the outpatient department before pneumonia developed. Necropsy revealed the sigmoid sinus and jugular vein perfectly healed. It was not felt that the original infection could have been a factor.

REPORT OF CASE.

A boy, aged twelve years, was referred by his physician, because, suddenly on the seventh day in the course of acute otitis media, a high fever had developed and the patient had appeared very ill and complained of pain in the mandible which extended into the neck; he also complained of trismus. There was no pain or tenderness over the mastoid.

Examination showed the temperature to be 105.5° F., pulse 115, and respirations 26. There was marked trismus on the left side. The left ear was discharging copious amounts of pus although there was no tenderness over the mastoid region. Other than for these observations, the ear, nose and throat appeared normal. The general examination was negative except for a soft systolic murmur. The neurologic examination disclosed slight rigidity of the neck, normal spinal fluid, and negative Tobey-Ayer test. The urinalysis was negative, the Wassermann reaction of the blood was negative, the hemoglobin was 65 per cent, erythrocytes numbered 3,750,000, leucocytes numbered 8,500, of which 85 per cent were neutrophils, and repeated cultures of the blood gave negative results.

Roentgenograms revealed a large diploic mastoid without evidence of breaking down. The zygomatic cells were markedly developed. It was felt that the zygomatic abscess would account for the pain and trismus and that a hemorrhagic type of mastoiditis and phlebitis of the sigmoid sinus would account for the clinical picture.

At operation, September 24, 1930, the hemorrhagic type of mastoiditis was found, together with a zygomatic subperiosteal abscess, but no physical changes of the sigmoid sinus were found. The sinus was purposely uncovered throughout its extent. There were no signs of thrombosis. The clinical course was not favorably affected. Two days after operation, respiration rose to 40. There was no cough and examination of the thorax was negative. September 28th, operation revealed thrombosis of the jugular bulb, and the jugular vein was found collapsed above the facial vein. It was divided between two ligatures. Bleeding was obtained from both ends of the sinus after the removal of the thrombus from the bulb. The next day the patient complained of abdominal pain, especially marked in the epigastrium, without a point of maximal tenderness. The clinical course was not improved. The temperature was of a septic type and the patient lost strength gradually. October 6th, transfusion of 250 cc. of blood by the citrate method seemed to be followed by some general improvement. The patient's appetite improved and he showed signs of cheerfulness. The temperature was no longer septic and respiration became more normal. October 16th, there were no further changes, and although the convalescence was not entirely satisfactory the patient seemed to be improving slightly. October 22d, at 3:00 a. m., the patient coughed once and raised 250 cc. of foul-smelling pus; his temperature dropped to normal and he was greatly improved. The pain in the epigastrium became less, and a roentgenographic examination following a barium meal was negative. From this time on, except for a rise in temperature on two successive days, convalescence was satisfactory. The patient's strength returned and the lost weight was regained. The parents reported that he has been normal in every way.

The primary thrombosis of the jugular bulb in this case may have been present at the time of the first operation, but if it was, the negative signs were misinterpreted. At the second operation the sinus appeared very different and was obviously affected toward the bulb. The condition of the thorax was now the question of greatest importance, as the symptoms all pointed to the thorax, although general examination was repeatedly negative. The parents said the boy had had pneumonia several years previously, at which time he was extremely ill. There were four conditions which were considered in the differential diagnosis: mediastinitis, pulmonary abscess, interlobar empyema and subphrenic abscess.

The abdominal tenderness without a point of maximal intensity was the outstanding symptom. This was accompanied at first by nausea and vomiting. Repeated general and roentgenographic examinations were negative. The abdominal tenderness was suggestive of some disturbance near the diaphragm. Mediastinitis without substernal pain, cough, some degree of dysphagia or dyspnea, or widening of the shadow in the roentgenogram, did not seem probable. It would seem that pulmonary abscess, interlobar empyema or subphrenic abscess containing 250 cc. of pus could have been made out. It is proverbial, however, that such a condition could exist along the spine in the so-called gutter without evidence of its presence. Absence of pain on deep breathing was a confusing sign. The examination with lipiodol was not used because of the extreme illness and lack of co-operation on the part of the patient. Subsequent study of a series of roentgenograms led the roentgenologist to state that the most probable diagnosis was subphrenic abscess, although pulmonary abscess or interlobar empyema could not be ruled out. In consultation it was suggested that the condition probably was pulmonary abscess, because, after the spontaneous evacuation, recovery took place. Subphrenic abscess has a much more protracted course and usually it is necessary to establish drainage.

SUMMARY.

Pulmonary complications occurred in a surprisingly low percentage of cases (42 per cent) in a series of 933 cases. There was no operative mortality in this group if the case in which pneumonia and empyema developed, after the mastoid wound had practically healed, is eliminated. The differential diagnosis in certain suppurative lesions, depending on the site of the lesion, may be difficult or impossible. Spontaneous rupture of an intrathoracic abscess with recovery is possible, as in the reported case.

MAYO CLINIC.

VII.

A CASE OF BRAIN TUMOR WITH OTONEUROLOGIC FINDINGS AND AUTOPSY.*

ALFRED LEWY, M. D.,

CHICAGO.

This case is reported for two reasons: (1) The otoneurologic findings alone were somewhat misleading as to localization; (2) otoneurologic tests followed by autopsy may be instructive.

O. R., male, age 63, married; three children. Three years ago patient had urinary incontinence at times and occasionally incontinence of the rectal sphincter when bowels were loose, from both of which disturbances he recovered.

Three months ago present ailment began with weakness in the left hand, extending to arm; then in left foot, gradually extending to entire left side. One week ago, on admission to the County Hospital, hemiplegia was complete. He has lost 30 pounds in weight in three months. The facial paralysis is less pronounced than it was eight weeks ago. (At present limited to mouth.) There is difficulty in swallowing and articulation at times. Other points mentioned in the history are buzzing in ears; diplopia, three months; dizzy spells at intervals, three months.

Examination on admission:

Left hemiplegia, the facial being paretic in only the lower part; right external rectus muscle paralyzed. Spinal puncture obtained only ten drops of fluid under low pressure, not increased by jugular compression; cells 4 per c. mm.; Wassermann negative.

Clinical diagnosis by neurologic department, "Tumor right side of pons."

Referred to otologic department about ten days before exitus. Findings at that time:

Tongue deviates to right.

*Read at meeting of Middle Section, American Laryngological, Rhinological and Otological Society, Ann Arbor, January 17, 1931.

Mucous membrane covering superior constrictor does not show movement of right side during phonation.

Deafness of marked degree, of inner ear type.

No spontaneous nystagmus; no head movement nystagmus.

Spontaneous past pointing with right hand to right; left not tested on account of paralysis.

Cold irrigation left; slow movement to left; quick component abortive (in both eyes) (right rectus paresis); appears in 30 seconds; past pointing to left with right hand (left not tested).

Cold irrigation right: horizontal and rotatory nystagmus to left (both components normal).

Past pointing with right hand to right (increased over the spontaneous past pointing).

Vasomotor disturbance marked; patient felt nauseated and weak.

On account of the patient's condition it was not practicable to go further with the tests at that time. The interesting features were that in spite of a lesion in the pons the impulses from the labyrinths went through, and the patient was hypersensitive in his reactions to the tests. The abnormal response to irrigation of the left ear was explainable on the basis of the right rectus paresis. That is, the quick component was abortive and appeared to affect both eyes. Question: Where is the center that correlates the combined movement of the recti for the quick component, and where the tract? According to Fisher, the caloric tests are not likely to produce vasomotor disturbance and nausea in subtentorial tumors. In this case, while the otoneurologic tests might be construed to suggest the possibility of a supratentorial lesion, the ordinary focal signs of a left hemiplegia with paralysis of several cranial nerves on the right side pointed to a lesion of the right side of the pons.

Autopsy revealed tumor of the right lower half of the pons. Diagnosis by Dr. R. H. Jaffe of the County Hospital as a spongioblastic neuro-epithelioma. Dr. Percival Bailey of the University of Chicago also examined sections and diagnosed it as a melanotic blastoma.

Herewith sagittal section of hind-brain showing part of the tumor.

Further pathologic findings were: Confluent bronchopneumonia, both lower lobes; gangrene, left lobe; fibrocaseous tuberculosis, both upper lobes; serofibrinous pleurisy, left; brown atrophy and passive congestion liver; infectious soft spleen; cloudy swelling kidneys; median bar hypertrophy of prostate; trabeculated bladder, catarrhal cystitis; colitis membranacea; nodose goiter; atrophy and degeneration of myocardium; decubitus sacral region.

25 EAST WASHINGTON STREET.

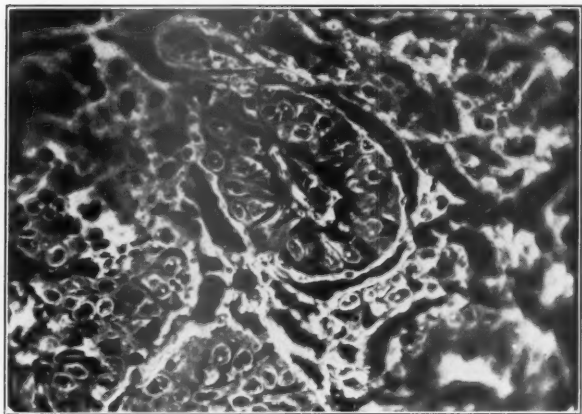


Fig. 1. Low power, neural tube; blood vessel in center, surrounded by epithelial cells and these by epiblastic membrane.



Tumor.

Fig. 2. Sagittal section of hind-brain, showing part of tumor.

VIII.

VITAMINS AND DEFICIENCY DIET IN THEIR RELATION TO SINUSITIS: ABSTRACT OF RECENT LITERATURE.*

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NEW YORK.

The last thirty years have witnessed a remarkable change in the treatment of disease by the medical profession. Thirty years ago the meetings of this Section and of those of our national special societies were given over largely to the description of new operations and the report of cases operated upon. Today scientific medicine is more and more concerned with the consideration of the prevention of disease. Fewer and fewer operations are reported. Among the pioneers who stressed the importance of preventive measures, so far as the nose, throat and ear are concerned, is J. A. Stucky of Lexington, Ky. In season and out of season, through all these years, he has been as the voice of one crying in the wilderness, proclaiming on every occasion which presented itself the close relationship between faulty diet and diseases of the nose and ear. No less earnestly and persistently has our fellow member, Dr. Coffin, dwelt on this relationship. The teachings of Stucky and Coffin required, however, the discovery of the vitamins to bring its importance home to the members of our specialty.

In 1918, Dr. Lee Wallace Dean carried out, at the University of Iowa, certain experiments in conjunction with Dr. Amy Daniels which demonstrated the close association between sinus infection and diet deficient in fat soluble vitamin A. Since that time much labor has been bestowed and many papers have appeared on the subject. Most recently the subject was discussed at the 1929 meeting of the American Laryngological Association in a symposium on *Nutritional Disturbances in Relation to the Nose*, engaged in by R. Adams Dutcher, Roy A. Barlow, Lee

*Read before the Section of Laryngology, New York Academy of Medicine, November 21, 1930.

Wallace Dean and Burt R. Shurly. Of these papers, that by Professor Dutcher on *Vitamins in Human and Animal Nutrition* is largely a history of the various vitamins and a discussion of their functions. In it he states that the first piece of work in the study of accessory food factors was done by Eijkmann (1897), a Dutch physician, who produced experimental beriberi by feeding polished rice to poultry. The name vitamin was devised by Dr. Casimir Funk in 1910 to 1912. Osborne and Mendel at Yale, and McCollum and his coworkers at Wisconsin discovered the existence of a fat soluble food factor in cod liver oil, butter fat and egg yolk which they named fat soluble A and a water soluble factor in rice polishings and in yeast which they named "water soluble B." Vitamin B is the antineuritic factor, vitamin C the antiscorbutic factor, and vitamin D the antirachitic factor. A deficiency in vitamin A causes a loss in appetite, loss in body weight and infections, especially in the sinuses, ears and lungs.

Barlow's paper on *Diet in Connection with Diseases of the Lymphatic System, Especially of the Upper Air Tract*, only indirectly has to do with diseases of the nose and accessory nasal sinuses. Barlow affirms that it is well known and generally accepted that diet free of vitamin A predisposes to sinusitis, bronchitis and pneumonia. In the light of the experiments which he carried out, Barlow is of the opinion that a diet defective in vitamin has little or no effect in itself upon the condition of the lymphatic system. It would appear that if any one element has a particular reaction on the lymphatic system it is fat, and this can be made more so by the deprivation of water. According to Barlow, so far nothing definite has been determined about the effect of diet upon the respiratory lymphatics. The weight of data rather points to the probability of fat being of clinical value, whether fat soluble or otherwise. Vitamins themselves, in his opinion, probably are not responsible for lymphatic changes, except vitamin A, and it is difficult to say how much of the lymphatic enlargement is due to vitamin A deficiency and how much to secondary infection.

Shurly's paper is on *Deficiency Diet in Relation to the Skeleton, Especially in Connection with the Bone Affections of the Head*. In his opinion, experiment, clinical findings and the laboratory

of nutrition in particular prove that the bony affections of the head are profoundly influenced by the supply of vitamins, especially A, C and D, and the ratio of phosphorus and calcium. He insists that if the bony defects of the head are to be remedied and prevented it is necessary for the otolaryngologist to join the pediatricist and orthopedist in an application of the newer therapeutic principles that are available in this field. The value of cod liver oil with the constituents of vitamins A and D is a well established therapy. As the result of his study, Shurly draws the following conclusion:

1. The structure, stability and growth of the bones of the head are dependent upon the supply of vitamin A, C and D, particularly with a determined ratio of calcium and phosphorus.

2. The prevention of rickets is a prenatal problem, together with the diet of the nursing mother. The value of cod liver oil, milk, eggs and leafy vegetables needs wider application.

Of all the papers of the Symposium the one that has the greatest interest for us is that by Dean on *The Relation of Diet to Diseases of the Sinuses*. As previously stated, as far back as 1918, Dr. Amy Daniels demonstrated the relation between sinus infections and diets deficient in fat soluble vitamin A. In view of this relationship, Dr. Daniels, in collaboration with Dr. Dean, sought to ascertain whether *established* sinus disease could be favorably influenced or cured by diet alone. Their investigations demonstrated that diet alone was inadequate and that in all cases of chronic sinusitis proper drainage was essential for a cure. Indeed, drainage with ordinary diet was more effective than a vitamin A diet without drainage. While this applies to chronic sinusitis, Dean and Daniels demonstrated clinically that cases of incipient nasal infections treated with a diet rich in vitamin A can be favorably impressed if administered with orange juice and a large amount of vitamin B secured from wheat kernels. These investigations strongly set forth the value of proper diet in sinus disease, especially of children, in connection with suitable local measures and proper hygienic conditions. While these observations apply particularly to children, Dean believes they are true in a large degree of adults and teach the lesson that in the treatment of most sinus cases proper diet is always essential.

Dean refers to the laboratory work of Daniels with white rats; in all of the rats studied she produced a suppurative sinusitis by administering a diet deficient in vitamin A. In twenty-five rats on normal diet examined as control, five showed a little reddening of the nasal mucosa. The other twenty were entirely free from any signs of mastoid or nasal sinus disease. The rats fed on a vitamin A deficiency diet all revealed on autopsy pus in their accessory sinuses and mastoids. Dean makes the interesting observation that at one time he established a normal infant ward to study the growing infant. Infants were placed there for two years. They received proper diet and were kept under ideal hygienic surroundings. During the two years neither a laryngologist nor an otologist was summoned to the ward. The cases were followed up after they left the hospital and only one developed mastoiditis, and in this case the mother had violated her instructions by mixing babies' milk with cod liver oil. Dean concludes his paper by declaiming that a deficiency diet does not cause infection; it only serves to lower resistance and lessen immunity. In the treatment of sinus infections a diet rich in vitamin A together with vitamin B is essential, although it does not take the place of either the proper laryngologic procedures or the observance of the usual hygienic regulation.

The latest contribution of Stucky on this subject was a paper read before the American Laryngological, Rhinological and Otolological Society at its 1924 meeting, entitled "Deficient Vitamin Diets as a Factor in Otolaryngological Conditions." Stucky, in this paper, raises the important question how much sickness is avoidable, and then stresses the importance of a proper diet in the prevention of disease. Our bodies are the products of the food we eat. Research workers have shown by experimentation and observation that the protective foods are the natural whole grain, milk, eggs, roots, greens, nuts and fruits used as soon as may be after cooking or not cooked at all, the most important of these being *milk, vegetables, a leafy salad and fruit*. Besides the proper vitamins two other important factors must enter into a normal dietary, mineral salts and cellulose or waste. If to whole grains containing mineral salts and roughage and leafy vegetables and fruits there be added milk, eggs and butter, an ideal,

properly balanced diet is obtained. The term "deficiency disease" represents disorders due to faulty or ill balanced food.

Stucky quotes the experiments with cats of Clarge of the University of Georgia, who was able to cause the increase or decrease in the size of tonsils and adenoids by the particular diet that he administered. As a result of fifteen years of observation among the people of the mountains of Kentucky, Stucky declares that many ophthalmologic and otolaryngologic diseases are local manifestations of a systemic disorder, the chief factor of this being in the intestinal canal. Highly milled cereals, potatoes, cane sugar and muscled meats have proven a failure in animal experiments and are proving a failure in human experience. Mineral salts is absolutely essential to the continued development and health of man. The diet which Stucky is in the habit of using is the same as that used by Daniels and consists of one quart of milk a day, one egg, whole wheat bread for at least two meals a day, butter, three vegetables, not potatoes, cereals, three fruits, one of which is raw, besides meats for the older children. If there are any signs of a vitamin A deficiency, one or two teaspoons of cod liver oil daily are added. He points out that when he first began his trip into the mountains of Kentucky, sixteen years ago, there was almost a complete absence of milk, eggs and butter in the diet of the natives. Their diet consisted largely of fried foods, coffee, pork and occasionally fresh meat. A suprising number of cases of suppurative mastoiditis requiring operation was met with. As soon as the children were placed in a settlement school, where they could get a well balanced diet, a decrease in the quantity of lymphoid tissue with fewer cases of discharging ears was noted. Experience soon taught him that the entire problem having to do with the eyes, ears, noses and throats of children could be solved by the physician, nurse and school teacher working together. He closes by saying: I am still convinced from my clinical observations that the endocrine imbalance and decrease in the alkali reserve of the average case is due to improper feeding. In some schools and orphanages he has been able to prevent the usual postfestival epidemic of sore throats, nose and ears by carefully regulating the diet of the children during the seasons, keeping them away from the usual overindulgence of these fes-

tival occasions. In the discussion which followed, Dr. Dean referred to his practice of insisting on proper feeding in his hospital wards and was able to demonstrate to his own satisfaction, time and time again, that patients with acute infections of the paranasal sinuses and ear will make improvement without definite surgical or medical procedures—simply by additions to dietetic regime.

Another paper by Shurly to which we wish to refer is entitled "Vitamins and Dietetics in Relation to Otolaryngology," which appeared in the *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, March, 1928. Shurly sets forth here, just as Stucky has done, the importance of the study of the problems of infection and immunity and the prevention of the ravages of suppuration. Calling attention to the fact that 70 per cent of all focal infection is in the tonsils, sinuses and teeth, he stresses the rôle that biochemistry has assumed in the solution of these problems. The realm of dietetics in otolaryngology can be given no greater emphasis than in guarding the tonsil and adenoid child with marked lymphatism in its development, especially after operation. Shurly regards raw milk as the most satisfactory article of food taken by man. Caries of the teeth and pyorrhea to a large measure are the result of deficient diet. Research in the field of nutrition applied to preventive otolaryngology offers an opportunity to raise the vitality of all mankind. The errors of diet may not cause deficiency disease, but insidiously their gradual operation, more or less constant, may be responsible for undermining the resistance in a degree so masked that the menace is quite unappreciated. If we are to have well nourished tissue in the nose and throat, we must have a well balanced basal metabolism. The popular conception that any diet which complies with the composition of ordinary combinations affords and furnishes protein and energy in palatable and digestible form, would promote growth and maintain health and long life is destined to pass into the background. The experiments conducted under his direction with white rats in Detroit confirm the findings of other observers, especially those of Daniels, that diet deficient in vitamin A which is plentiful in butter fat, egg yolk, cod liver oil and the fats of liver and kidney, exhibits on the 28th day a lowered resistance to infection. The rats thus treated show a well developed nasal

sinus infection and also extension to the middle ear. The controls in great numbers fail to develop suppuration. He concludes by saying that while the research laboratory alone will be able to furnish a final solution to the interesting relationship that exists between food and pathology in the ear, nose and throat, and elsewhere, we may well believe that a genuine practical application of dietetics and therapeutics is indicated in otolaryngology together with our surgery. The demand of the human economy for iron, phosphorus, calcium, iodine and vitamins is present in otolaryngologic pathology more frequently than in other deficient parts of the body. Our rôle and our influence in the realm of pediatric otolaryngology is most extensive, as malnutrition, adenitis and infection are more prevalent in our domain.

Finally, in line with the paper just considered, Shurly, at the meeting of the Medical Association in Portland, in 1929, read a paper on the same subject entitled "Infection of Accessory Sinuses and Upper Respiratory Tract in Vitamin A Deficiency." In this paper he reports the results of a bacteriologic study of the subject he had made, based on the generally accepted recognition of a pathologic entity known as xerophthalmia, the result of deficiency vitamin A diet, evidenced by lowered resistance to infection and the development of lesions in the lacrimal apparatus, corneal ulceration and of infection of the nasal passages, accessory sinuses, middle ear, tongue and upper respiratory tract. According to Shurly, we are to look to biochemistry to explain why acute sinusitis and mastoiditis prevail in epidemic form and change in type overnight. Prompted by this view, Shurly, in conjunction with R. G. Turner, conducted a laboratory study of the pathogenicity of certain bacterial organisms isolated from infections and suppuration of the nasal cavities and middle ear in albino rats deprived of vitamin A. He states in his paper the results of his investigations and from them draws the conclusions that organisms isolated from suppurations of the upper respiratory tract and middle ear in albino rats suffering from lack of vitamin A, morphologically appearing as gram-negative cocci, will produce a fatal toxemia in rabbits. From the observations made, it appears that the poisonous effect is produced by an endotoxin and not by a toxic substance secreted by the organism.

IX.

DIET IN SINUSITIS.*

LEWIS A. COFFIN, M. D.,

NEW YORK.

There has been during the past fifteen or twenty years a wonderful advance in the scientific knowledge of foods. There can be no question as to the existence and actions of the better known vitamins, as there is a wonderful agreement among the various scientific workers the world over.

Dr. Harris speaks of the ever decreasing number of operations described and reported, and says that today scientific medicine is more and more concerned with the consideration of the prevention of disease. This is undoubtedly true, but let nobody think that fewer operations are done or that fewer conditions demanding operation exist.

Occasionally in various parts of the world conditions of life are such as Stucky found in the mountains of Kentucky, where any changes for the better as to cleanliness and scientific dieting must make for better health, better resistance to disease, and fewer operations. But, as to the average individual, living the average life of our American citizens, I doubt if as yet scientific medicine has done much in the prevention of such diseases or conditions as we as laryngologists are called upon to operate on and treat. Much money and labor are being expended that this end may be brought about.

There seems to be no danger in the use of vitamins so long as we confine ourselves to giving them in the food in which they naturally occur. Vitamin D, obtained from the irradiation of ergosterol, seems not so innocent. Deaths have been reported from its use—the autopsies of two children, at least, showing

*Read before the Section of Laryngology, New York Academy of Medicine, November 21, 1930.

that they had died from calcification of the kidneys and various lymphatic glands. I have known of one of our leading pediatricians withdrawing it from the diet of children under his care because it had come to his notice that its use had led to arteriosclerosis in some of the laboratory rats to which it had been fed. Vitamin D, as appearing in viosterol, would seem to owe its physical and chemical constitution to a latent radiant energy derived from ultraviolet ray. Such a body under certain circumstances must give up this energy. The when and where that this occurs must be of considerable moment. Much credit is due to the pediatricians for the splendid work they have done in the artificial feeding of children of the present day. For the past twenty years it has been the fashion for the young women of our land, the prospective mothers of future generations, to abstain from fattening foods. This diet must be of the nature of a vitamin A deficiency type, and that to the extent that the offspring during gestation is deprived of vitamin A. After birth the mother is seldom able to furnish the child with proper nourishment. That most beautiful picture of human relations, the mother and nursing child, seems to be a passing one.

The pediatrician has met the issue and is so able to artificially feed the babies today that some consider that as a class they are healthier and more robust than ever. Much credit for this condition is undoubtedly due to our welfare workers.

Dr. Harris has justly paid high tribute to Stucky. Stucky, like some other laryngologic workers, early in his career was struck by the frequency with which rhinologic disorders were associated with gastrointestinal disorders. So consistently was it found that certain rhinologic disorders were found to be associated with certain gastrointestinal disorders that the rhinologist came to feel that he could diagnose the gastrointestinal disorders from the nasal findings. The treatment of the diseases of the stomach and bowels has led to the study of diet by many rhinologists, and I trust that in the near future we may have to discuss an abstract of literature on a subject worded something like this: "Dietary Factors Other Than Vitamins as Affecting the Upper Respiratory Tract."

Dr. Dutcher, who read the first paper of the Symposium to which Dr. Harris has called our attention, gives this advice:

"Do not worry about development of deficiency diseases. Feed sufficiently varied diets to prevent lowered resistance and susceptibility to other diseases," and advises us that "it is generally possible and preferable to obtain our vitamin supply from the dairy, the garden, the orchard and the poultry yard."

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X.

DIET IN SINUSITIS IN CHILDREN.*

ADOLPH G. DE SANCTIS, M. D.,†

NEW YORK.

Sinusitis in children has become a problem of paramount importance because of its increasing prevalence, the difficulties encountered in diagnosis and especially because of its resistance to treatment. For the past decade many investigators have attempted to establish a definite relationship between dietary disturbances and sinus disease. At various times, various workers have ascribed the etiology to deficiencies of the various vitamins A, B, C and D, and to deficiencies of the food elements themselves. Let us for a moment consider the vitamins.

It has been rather definitely established that vitamin A promotes the function of tissues, and it is more or less certain that appetite and growth are dependent on this vitamin. Experimentally in rats, a deficiency of vitamin A causes loss of appetite, loss of body weight, susceptibility to infection, especially of the eyes, ears, sinuses, air passages and probably lungs. The mucosa of the respiratory tract becomes of the stratified squamous type. Whether a deficiency of this same vitamin has the same significance in humans as it has in rats is still a matter of speculation. It is well to remember that until five years ago vitamin A was believed to be the antirachitic factor. Then this theory was abandoned in favor of vitamin D as the antirachitic factor. I am not at all convinced, however, that vitamin D alone is responsible for rickets. We were able to show and report that, although irradiated ergosterol 100 D. given in quantities to supply twice the number of rat units of vitamin D as that of cod liver oil, the latter prevented rickets in 97 per cent of the cases, whereas irra-

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†From the Pediatric Department of the New York Post Graduate Hospital.

diated ergosterol prevented it in 73 per cent of cases. My conclusion at the time was that rickets was not due alone to a deficiency of vitamin D but that vitamin A may be also a factor. I feel that it is well to emphasize that viosterol is not cod liver oil and never will be. Whether the new 250D viosterol will prove to be as valuable an antirachitic as cod liver oil is still a question.

Lymphatic glands are actually concerned with the arresting and walling off of infection, and if an otherwise healthy gland is altered by dietary disturbances, knowledge of this fact will enable us to aid nature in combating many manifestations of respiratory infection.

Rats fed on vitamin A free diet soon develop a marked conjunctivitis, watery secretions from the nose and often corneal ulcers. The bronchial lymphatics on autopsy, however, show very little demonstrable change in these animals. This latter fact does not support the popular and generally accepted theory that a diet free of vitamin A is directly and intimately related to infection of the upper respiratory passages.

Animals fed on diets deficient in the antineuritic factor vitamin B and antiscorbutic factor vitamin C show no changes whatsoever in the lymphatics of the upper air passages. Vitamins B and C are essential to normal growth and appetite, and therefore necessary in the diet of the growing child. They, however, have no direct relationship to infection of the upper air passages except in so far as all children with lowered resistance as a result of avitaminosis are more susceptible to disease. A vitamin D free diet produces in rats the characteristic rachitic changes, but again no change in the lymphatics. It is commonly known that rachitic infants and children are more prone to upper respiratory disease than the normal nonrachitic. But as with vitamins B and C, the relationship to sinus disease is only an indirect one, again that of lowered body resistance. The same is true of vitamins E and G, the antisterility and pellagra preventing factors, respectively. It might therefore be properly said that a diet deficient in any vitamin (except vitamin A) has little or no effect upon the lymphatic system. Of the vitamins studied, however, there can be little doubt that vitamin A deficiency probably renders the

lymph glands susceptible to infection and bacterial invasion. Up to a short time ago it was impossible to obtain a concentrated vitamin A preparation. With the aid and co-operation of the experimental laboratory of one of our American manufacturers, we have now a concentrated vitamin A, a preparation of alfalfa extract in olive oil, having four times the vitamin A potency of that of cod liver oil. We have been using this preparation in chronic respiratory infections in children for a short time, but it is much too early to report on its value. It will prove very interesting to observe the results in those cases of sinus disease in children receiving this concentrated vitamin A preparation and compare them with those receiving the same local treatment and diet but without the extra concentrated supply of vitamin A. Such a study is now under way. Cod liver oil, the usual supply of vitamins A and D, very often for one reason or another cannot be tolerated by the patient for any length of time, and I, for one, certainly have looked with favor upon the recent introduction of more palatable forms of A and D vitamins. At present, there are available two concentrated preparations, one in an oil base and one in tablet form.

Of the food elements themselves, besides vitamins, the one that seems to be more or less related to respiratory infection in general, is fat. The consensus of opinion is that sugar and protein do not seem to alter the lymphatics and therefore play little if any part in the cause of respiratory infections. Although in regard to protein this may be true of adults, I feel that growing children will do much better on high protein diet in the treatment of sinus disease. Children naturally require more protein to allow for growth as well as to repair body waste. Although it is time honored therapy to force fluids in the treatment of acute respiratory conditions, I do not believe that restriction of fluids in the diet per se plays an important part in the etiology.

After this brief discussion of diet in general, we must note that whatever the relationship is between dietary disturbance on one hand and sinus disease on the other, it surely must be only an indirect one, probably one of lowered resistance of the upper air passages to infection. For if it were not so, a proper diet treatment, rich in all vitamins, would be either a specific or at

least more successful than it really is. The diet which we recommend should contain a liberal amount of butter, vegetables, fruits, milk, eggs and cereal foods. So far as possible, all fat should be supplied in the form of butter or cream; vegetables should be given at least twice a day, and fruits, especially oranges, should be used freely, since these furnish the antineuritic vitamins necessary for appetite stimulation. The amount of carbohydrate foods should be limited, for there is a tendency to use too much bread, potatoes and cereals in the diet of most children. To be certain that there is a liberal amount of vitamin A and vitamin D being supplied, cod liver oil as such or one of its concentrates is also given. This really constitutes a high fat and high protein and comparatively low carbohydrate diet, which is essentially the diet used in the treatment of pulmonary tuberculosis. Animal experimentation has definitely proven the value of this diet in combating tuberculous infection. The animals that were fed on a high fat and protein and comparatively low carbohydrate diet, when inoculated with tubercle bacilli developed a low grade chronic type of tuberculosis, whereas those fed on a high carbohydrate and low fat protein diet when inoculated with tubercle bacilli of equal virulence developed the acute form of tuberculosis, from which a number died. I believe that in the diet (whatever it may be, whether it be vitamin, chemical or just food) that is so successful in the treatment of tuberculosis are the same factors that are so helpful in the treatment of sinusitis and perhaps in other forms of chronic infection in children. For the past four years we have been using the high fat, high protein and low carbohydrate diet, with an added supply of vitamins A and D, in the dietetic management of children with sinusitis. A sample diet would contain:

1. One quart of milk daily.
2. Two eggs daily.
3. Plenty of vegetables.
4. Fruit juices and fruits.
5. One tablespoonful of butter with each meal.
6. Meat and fish once a day in children old enough to receive them.

7. And the addition of cod liver oil or a concentrated preparation of vitamins A and D.

The results with this diet were in some instances striking, particularly from the standpoint of weight and general condition. This effect was more noticeable in private patients than in dispensary patients. It is by no means to be assumed that the gain in weight is to be attributed to diet alone, as laryngologic procedures were carried out in the course of the disease, which I feel are equally important or even more essential in the treatment of sinusitis than diet. It is not uncommon to see children with sinus disease who, although receiving proper diets with the necessary vitamins, are not improving in general condition. Very often after proper treatment is instituted by the laryngologist, the improvement is striking and immediate. On the other hand, the reverse condition may be true. The patient then will not make satisfactory progress until the diet is corrected and proper hygienic measures advised.

After all, in the treatment of sinus infection, proper diet, though it plays an important part, cannot take the place of the proper local treatment. It is only through the proper co-operation of the laryngologist and the pediatrician that the best results may be obtained.

XI.

THE RELATION OF MASTOID STRUCTURE TO EXTENSION OF MASTOID INFECTION.*

BERNARD J. McMAHON, M. D.,

ST. LOUIS.

We are frequently puzzled by the lack of consistency in the behavior of infections of the middle ear and mastoid, and are prone to explain these variations principally by certain known peculiarities in the virulence and toxicity of the infecting organisms, with little thought given to the microscopic character of this bony structure or of the lining membrane of its cavities or of the intimate vascular relationship of the two. In order that we may have a proper perspective of all conditions, we should have a working knowledge of the histopathology involved.

It is therefore necessary for those of us who perhaps have a more active interest in the study of histopathology to try to present our findings in a manner easily and quickly grasped by the clinical otologist. Research fails in one of its most important purposes when it is not easily translatable and readily applied. Such a lack of coaptation will necessarily delay the progress of otology by continuing to discourage the potential interest of those who might otherwise be anxious to assume this knowledge.

Through the eustachian tube, the middle ear and mastoid antrum we enter into a more or less constant zone of connecting cells called the para-antral cells. These are usually of various sizes, an occasional one being as large as the antrum itself. Most of these are separated by bony septa, some of which are incomplete, while others have only a delimiting membrane reflected off the tips of incomplete septa and composed of but two layers of low epithelial cells, one layer from each side of the septum. The more peripherally located cells are also irregular in size and

*From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri. Read before the St. Louis Ear, Nose and Throat Club, February 18, 1931.

shape, but all connected, and the intervening bone of varying thicknesses.

The marrow spaces, large and small, apparently intercommunicate throughout. They have no lining membrane, but contain erythrocytes, erythroblasts, polymorphonuclear leucocytes, myelocytes (basophilic and eosinophilic) and giant cells or megakaryocytes. Within the body of the bone are small spaces which are not cells in the sense of being air containing, as they are filled with a loose, sparsely cellular areolar tissue in which there are one or more blood vessels, the sizes of which vary.

While there is no doubt that the size of the mastoid cells has some influence upon the rapidity of the spread of infection, it is the lining membrane (Fig. 1) of the bony cavities that is of paramount importance, and our knowledge of its functions cannot be too exact. The composition of this membrane is a tunica propria and a layer of surface epithelium. The tunica propria, or sub-epithelial tissue, consists of a rather loose areolar tissue groundwork, among the strands of which are found numbers of young connective tissue cells, some plasma cells and a few monocytes, but, of greater importance, a veritable network of small, thin-walled capillaries. (Fig. 2.) Some of these contain blood cells and endothelial cells, while others are probably lymph vessels, as they appear empty. This is the picture of the tunica propria in contact with the bone, a continuous system from the eustachian tube to the smallest bone space. It does not extend into the marrow spaces but only to the openings. As a consequence, these are closed off from the air containing cavities of the adjacent cells, though capillaries are seen to pass into the marrow spaces, and red and white blood cells from the marrow spaces are seen to cluster about capillaries within the tunica propria of the neighboring cells, thus establishing a close communication between the mastoid cells and the marrow spaces. The actual construction and cellular nature of normal tunica propria seems to be identical from fetal life to advanced age. (Figs. 2 and 3.)

The epithelial covering varies in character, according to the locality. That in the eustachian tube (Fig. 4) and the middle ear cavity below the level of a line between the orifice of the

eustachian tube and the lower margin of the oval window, is high columnar and ciliated, especially as one approaches the tubal orifice. (Figs. 5 and 6.) In the adjacent areas of the middle ear is found a lower columnar nonciliated type, while in approximately the upper half a single or multiple layered cuboidal or flat epithelium. This latter type also covers the tunica propria of the antrum, para-antral and all other air containing cells. (Figs. 1, 3, 7 and 8.)

The bone substance varies in character according to its proximity to the inner ear, that nearest called periotic and composed of the perichondrial, endochondral and intrachondral types, according to the classification of Bast, having a greater vascularity and a closer resemblance to cartilage, even in adults; that more distal being more dense and simulating bone elsewhere in the body, except that the Haversian systems are not as evident. The bone also differs according to the age of the individual—that is, the younger the bone, the more cartilaginous it is and the more numerous the cells, while the reverse is true in older bone. The vascular supply is extremely rich, in places showing a meshwork as close as that of the tunica propria with which its vessels are directly continuous. (Fig. 1.) The bone surface of the middle ear and antrum is not smooth but markedly irregular, due to juttings out of septa and depressions into open cells.

With the foregoing background of common thought as regards the mastoid structure, let us consider first the matter of the behavior of the mucosa of a normal mastoid, middle ear and eustachian tube, and then the response to an infection. The lining membrane has no glandular elements; therefore, it is non-secretory and needs no cilia to remove accumulated secretions, until the lower portion of the middle ear is reached. (Figs. 5 and 6.) Here it is that foreign matter or bacteria gaining access through the eustachian tube, or cell debris, is likely to accumulate, and from which it can be immediately ejected by the cilia in this region and in the eustachian tube. (Fig. 4.) There is probably some moisture in the middle ear, as there can be seen at intervals between the columnar epithelial cells transparent globules resembling goblet cells, which are most likely droplets of secretion passing to the surface from the tunica propria.

When infection occurs, there is first a marked edema of the tunica propria of the eustachian tube, then an infiltration with polymorphonuclear leucocytes, monocytes and red blood cells, the young connective tissue cells increase in numbers, and there are at intervals larger clusters of polymorphonuclear leucocytes around the capillaries. (Fig. 4.) The walls of the capillaries being composed only of a single layer of endothelial cells (Fig. 3), can easily be penetrated by the infecting organisms which can be carried by the blood and lymph streams much more quickly and easily farther along into the tunica propria of the middle ear, the antrum, the para-antral cells and the bone spaces than can take place by the direct route of free pus in the open cavities with no mechanism for driving this pus upward and backward. The tunica propria is so loosely woven that when infected an edema so extreme as to completely fill a cell long before the free pus can reach it, is easily conceivable, for we very often see a mastoid with isolated cells filled with pus and other intervening cells containing hyperplastic mucosa only. A similar edema can quickly occur in the attic and very effectually block off the aditus ad antrum. Therefore when we do encounter a cell filled with pus, it is very often a pus-laden and necrotic tunica propria and not an abscess in the sense of a similar condition in a purulent paranasal sinus disease. Obviously a combination of both conditions can and does exist in the mastoid, especially in the antrum and larger cells. (Fig. 7.)

The infection is carried into the bone in like manner, since the capillaries extend directly into it from the tunica propria. (Fig. 1.) Following bacterial invasion, the local cellular reaction in the bone spaces takes place, then the calcium is seen in the process of resorption, the bone cells either being destroyed or surviving and reassuming their original character of connective tissue cells, this tissue then becoming infiltrated with polymorphonuclear leucocytes and bacteria, and fusing with an adjacent abscess cavity. This process can also be seen taking place along the bone edges underneath the tunica propria of the air cells, the endosteum being infiltrated and thickened. (Fig. 8.) This absorption of calcium as a result of infection may well explain the indistinct cell outlines in the roentgenograms of infected mastoids. It would

seem as if the connective tissue cell which originally expressed its selectivity by attracting calcium and becoming a bone cell, repels calcium under the abnormal conditions of infection. Should it survive, it later again attracts calcium, which is deposited, not in the orderly manner of a septal arrangement but as a general mass action, or heteroplastically, thus establishing a sclerotic or eburnated type of bone structure, with few bone cells and widely separated and small bone spaces transmitting blood capillaries.

It is easily understandable, then, how we may have a mastoiditis without middle ear symptoms, on this theory of vascular and direct extension of infection along the tunica propria from the eustachian tube to the mastoid cells. As the tunica propria extends into the bone structure from the mastoid cells (Fig. 1), it likewise eventually reaches to the periosteum, and an infection following this course will give rise to a periostitis and palpable tenderness long before the superficial cells actually contain pus. The epithelial covering (Figs. 4, 5, 6 and 8) likewise undergoes the usual reaction of swelling of the cells and in some instances a later sloughing. This sloughing may be only of the superficial layer, due to a localized devitalization, or a more extensive fragmentation as a result of the rupture of an abscess of the sub-jacent tunica propria.

When we consider the extreme vascularity of the mucosa and bone of the mastoid, and the delicate walled type of vessels, an ideal medium for a rapid and extensive absorption of toxins, even from such a relatively small area, it is not difficult to realize why there is such a profound constitutional reaction to mastoid and middle ear infection, and why there is such a comparatively rapid recovery after the pus, the infected mucosa and bone have been removed and this vascular area completely eradicated. However, the rapidity of recovery is also dependent upon the extent of the involvement and the subsidence of the infection of the mucosa in the middle ear cavity. (Figs. 4 and 5.) It must be kept in mind that although the major portion of pathologic tissue has been removed, that in the middle ear must recover more slowly, under proper drainage through the aditus and the membrana tympani. The permanent damage to the tunica propria will then be in direct proportion to the number of connective tissue cells which

remain and mature at the site of invasion in the eustachian tube, about the walls and the ossicles in the middle ear cavity, and in the mastoid cells.

SUMMARY.

1. The eustachian tube, middle ear, mastoid antrum and bony cavities are lined with a richly vascular areolar tissue.

2. The mastoid bone substance is similarly vascularized in intimate connection with the areolar tissue vessels.

3. The greater evidence of infection is found microscopically about the vessels in the subepithelial areolar tissue.

4. The theory of extension of infection from the eustachian tube and middle ear by way of the vessels and substance of the subepithelial areolar tissue seems much more tenable than that of direct extension by way of the mastoid air cells alone.

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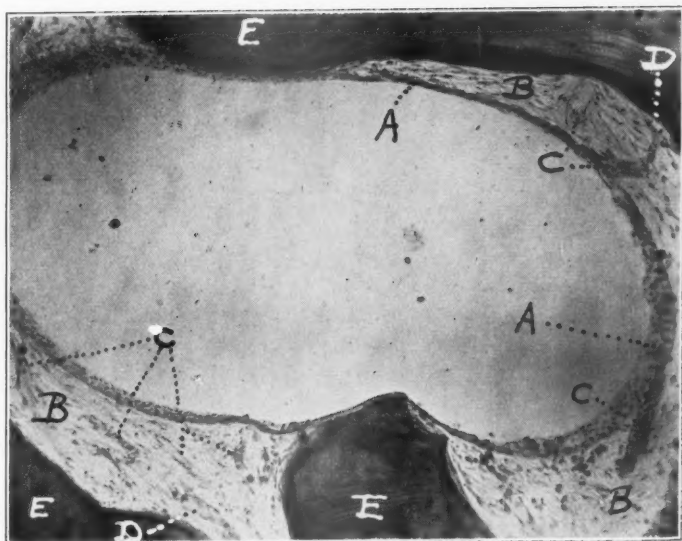


Fig. 1. Mastoid air cell normal. (Low power.) Age of patient 13 years.

A—Cuboidal epithelium.

B—Tunica propria.

C—Blood capillaries in tunica propria.

D—Blood capillaries extending into bone substance.

E—Bone.

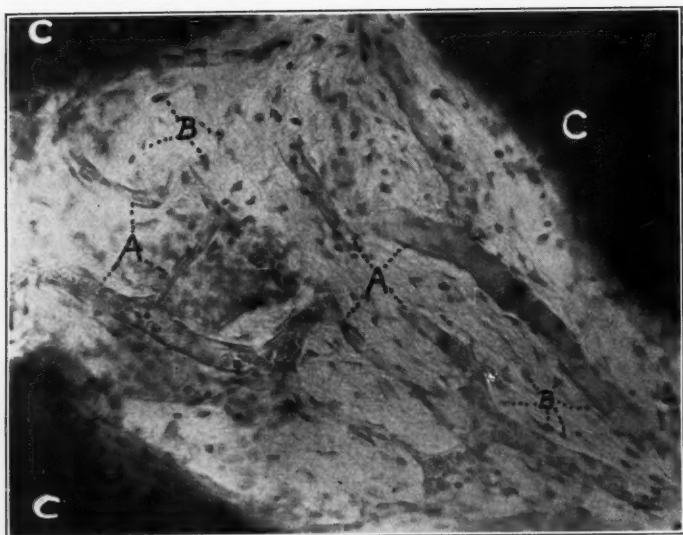


Fig. 2. Mastoid bone space normal. (High power.) Same patient as in Fig. 1, showing network of thin walled blood capillaries.

- A—Blood capillaries in tunica propria.
- B—Connective tissue cells of tunica propria.
- C—Bone.

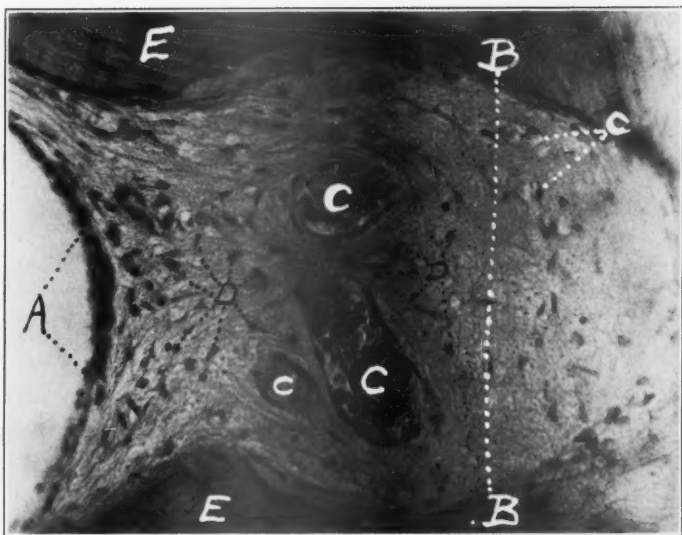


Fig. 3. Edge of mastoid air cell normal. (High power.) Age 59 years. Showing similarity of tunica propria to that in Fig. 1 and Fig. 2.
 A—Cuboidal epithelium.
 B-B—Tunica propria.
 C—Blood capillaries in tunica propria.
 D—Connective tissue cells in tunica propria.
 E—Bone.

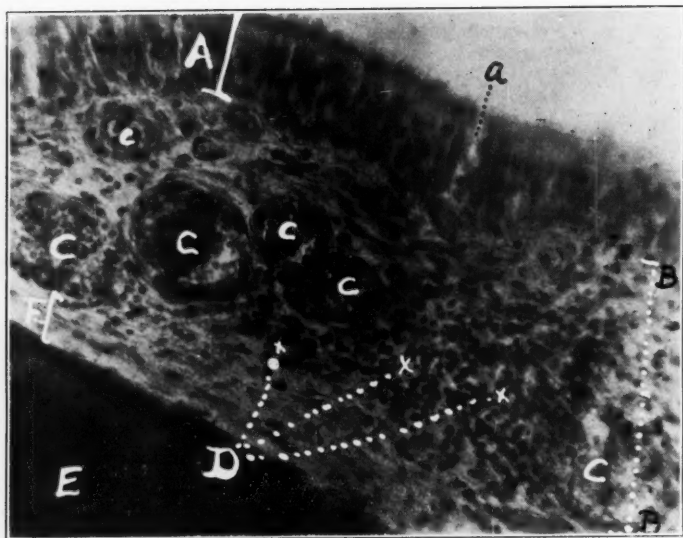


Fig. 4. Mucosa of eustachian tube, tympanic region. (High power.) Acute purulent otitis media for seven weeks before death. Age of patient 16 months.

- A—High columnar epithelium, ciliated, with beginning disintegration of individual cells (a).
- B-B—Tunica propria, edematous.
- C—Blood capillaries engorged.
- D—Polymorphonuclear leucocytes clustered about capillaries.
- E—Bone, normal.
- F—Endosteum thickened.

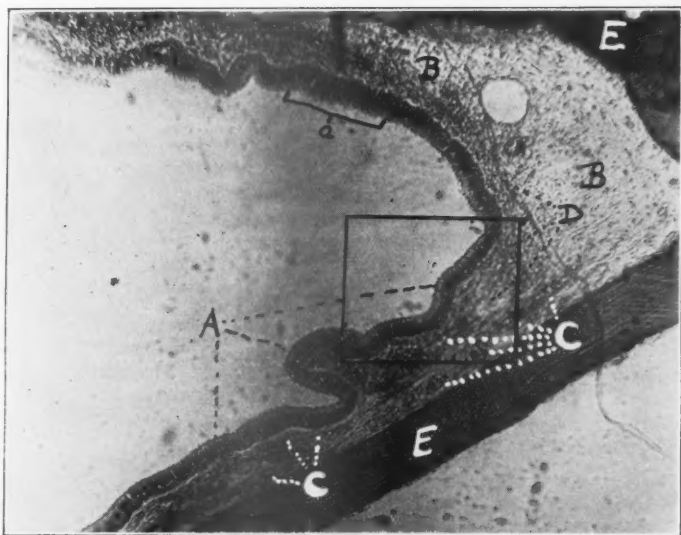


Fig. 5. Mucosa of middle ear, junction of floor with lateral wall. Same patient as in Figure 4; showing progress of infection. (Low power.)

A—High columnar epithelium, ciliated; beginning disintegration in places (a).

B—Tunica propria, edematous.

C—Blood capillaries, engorged.

D—Polymorphonuclear leucocytes with infiltration marked about capillaries and underneath epithelium.

E—Bone, normal.

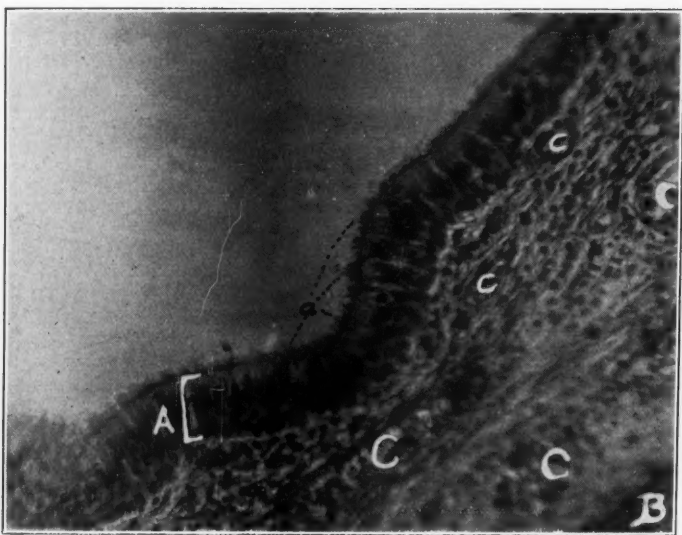


Fig. 6. High power magnification of area outlined in Fig. 5, showing detail of ciliated columnar epithelium and intensity of the perivascular polymorphonuclear leucocytic infiltration of the subepithelial tissue.

A—Epithelium. a-cilia.

B—Bone.

C—Blood capillaries.

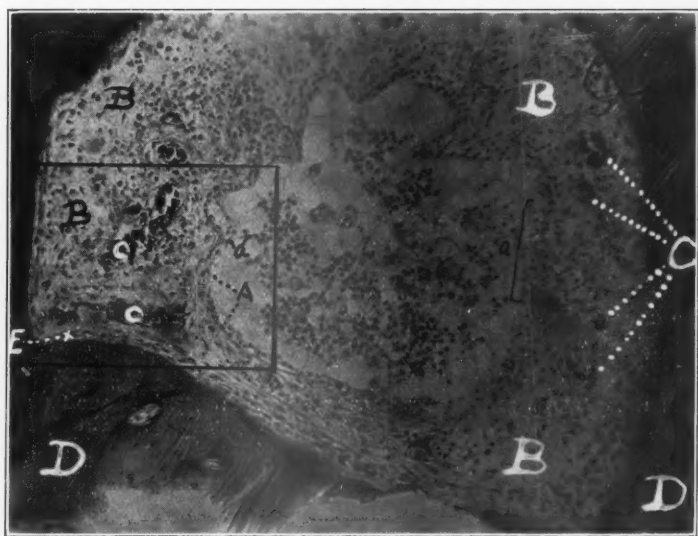


Fig. 7. Mastoid air cell, containing large numbers of polymorphonuclear leucocytes and desquamated epithelial cells; same patient as in Fig. 4. (Low power.)

- A—Cuboidal epithelium, intact; (a) disintegrated.
- B—Tunica propria extremely edematous and markedly infiltrated with polymorphonuclear leucocytes.
- C—Blood capillaries, engorged.
- D—Bone, normal.
- E—Endosteum, markedly thickened.

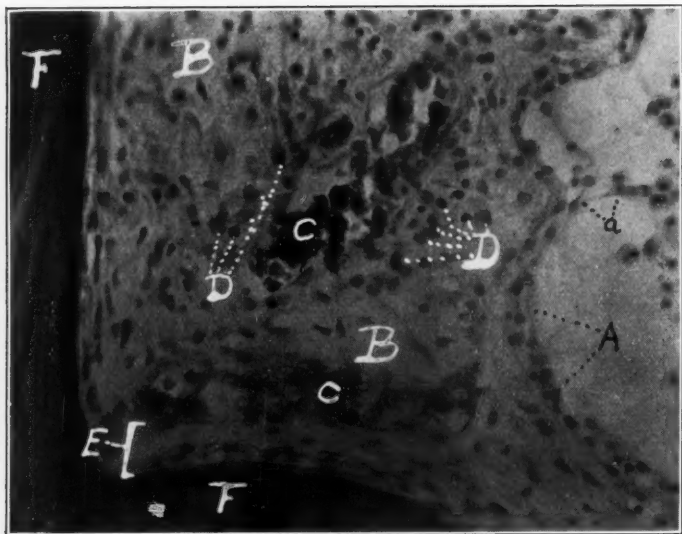


Fig. 8. High power magnification of area outlined in Fig. 7; showing the extreme thickening of the tunica propria due to the edema.

A—Cuboidal epithelium intact; (a) sloughed with free cells in air space.

B—Tunica propria, extremely edematous, marked increase in young connective tissue cells.

C—Blood capillaries, engorged.

D—Polymorphonuclear leucocytes, monocytes about blood capillary.

E—Endosteum markedly thickened.

F—Bone.

XII.

ALLERGY IN THE MIDDLE AND THE INTERNAL EAR.

ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

It is probable that no tissue of the body is free from specific reactions to substances foreign to the organism. Furthermore, it is characteristic of these reactions that they are not generally distributed but remain localized, although not necessarily affecting the same location in each attack. This being the case, the symptomatology depends less upon the nature of the reaction than upon its location and the specific tissues involved.

For this reason, the otologist will do well to bear in mind that allergy may simulate many diseases, both in appearance and symptom-complex. The literature at present yields little. Lewis,¹ in 1929, reported six cases of acute otitis media which he attributes to allergic influences, and his descriptions strongly indicate the soundness of his diagnosis.

Kobrak,² in a general review of the agencies which precipitate crises in hypersensitive individuals, devotes some pages to the ear. He cites hypersusceptibility to quinin and the tinnitus it produces; and attributes transient hypersensitiveness to noise to irritations of an allergic nature. "In the matter of true allergy of the eighth nerve," he says, "we arrive at unexplored scientific territory."

He describes a sudden severe attack of vertigo and nausea which seized one of his patients of known allergic antecedents; when this gentleman paid a visit to his goose farm. Unfortunately this was located in the provinces, so that while the history is extremely suggestive, Kobrak was unable to observe the attack himself or to make any immediate tests.

Duke³ reports two cases of so-called Ménière's symptom-complex, one of which improved on omission of vegetables the other of which was precipitated in a hay fever sufferer by the ingestion of spinach, vinegar and prunes.

The belief that allergic disease of the ear may prove relatively infrequent, once it is recognized, prompts the following descriptions of conditions, as observed in the labyrinth and the middle ear, in a still limited series of cases:

1. REACTIONS IN THE LABYRINTH.

In the few patients observed, allergic reactions in the labyrinth are essentially those of sudden vestibular irritation with inconstant cochlear disturbances. The patient is seized with attacks of vertigo of varying intensity, usually accompanied by some nystagmus, which is accentuated upon looking right or left. There is often a high-pitched tinnitus and a slight loss of hearing on the affected side, which may precede, accompany or follow the attacks.

The crises persist from one to forty-eight hours, during which the patient may experience a succession of transitory attacks of vertigo or may be totally incapacitated. In the intervals between attacks, the vestibular reactions are normal and the hearing may be unimpaired.

Kerrison² hints at their origin being extrinsic to the ear in a paragraph entitled "*Vertigo ab aure laeso*," in which he condemns the term as inappropriate. "A careful analysis of the symptoms described," he says, "shows that they coincide exactly with the usual phenomena of vestibular irritation, the one characteristic feature not invariably mentioned being nystagmus. In the light of recent advances in our knowledge, a close study of the symptoms will show these attacks to depend upon causes not easily traceable to a tympanic lesion, or at least to accord better with other theories of origin. . . . Each case should be subjected to the most careful observation and study, and an attempt made to determine exactly the underlying cause. '*Vertigo ab aure laeso*' is an inexact term which has supplied a convenient cloak for a gap in our knowledge."

Portmann⁵ describes the signs of vasomotor influences on the inner ear but does not associate allergy with the phenomena. He quotes Ferreri as having pointed out certain affections in the internal ear in connection with some sympathetic lesions in which he found a hypertension of the endolymphatic fluid. A portion

of Portmann's article is quoted here, as it contains a comprehensive list of tenable theories of the pathogeny of Ménière's syndrome:

"Escat, in a very complete work published in 1906, on arteriosclerosis of the labyrinth and of the acoustic centers, has devoted a large part to vascular spasms in the pathogeny of Ménière's disease. But it is especially Lermoyez who (in 1919) has insisted on the vascular labyrinthine phenomena and their clinical manifestations. He showed the importance of angiospasm of the vestibular or cochlear branches of the internal auditory artery in certain cases of deafness or vertigo.

"The first in date of these hypotheses was that of Ménière himself, viz., that the affection was caused by an intralabyrinthine hemorrhage; this, he said, had been proved at the necropsy of a young girl who had died after several severe attacks.

"Alexander, however, strongly attacks this opinion and advances the hypothesis that it is rather a question of a patient suffering from leukemia with leukemic hemorrhage in the labyrinth.

"Quincke, on his side, has stated his opinion that Ménière's disease is due to a phenomenon similar to that which causes the 'dermic circumscribed edema.' He thinks, no doubt, of an acute angioneurotic edema in the labyrinth, followed by an endolymphatic hypertension.

"According to Wittmaack, it is a question of intermittent variation of hypertension of the endolymphatic fluid, entailing modifications of the intralabyrinthine pressure. This author, relying on his experiments, thinks that the hypertension is due to a hyperplastic catarrhal state of the middle ear, with diffusion of toxic substances through the 'windows.'

"The Danish authors, S. H. Mygind and Hwidt, are of the opinion that the fits of vertigo are in connection with an intermittent increase of the endolymphatic pressure, but do not give any indication of the cause of this hypertension.

"Other authors, as Bárány, Aboulker, Quix, have thought rather of an intracranial origin. They see in a hypertension in the area of the lateral cisterna, causing a pressure on the eighth pair, and also on the medulla oblongata, the pons and the cerebellum, the cause of Ménière's vertigo.

"Then Quix asserts that this hyperpressure must act above all on the otolithic apparatus by the way of the 'ductus endolymphaticus.'

"S. H. Mygind makes a similar suggestion, while Thornval, who thinks also of an affection of the otolithic system, places the original lesion in the medulla oblongata.

"Kobrak ascribes the pathogeny of Ménière's vertigo to angioneurotic fits of the eighth pair of cranial nerves, depending on two principal causes: (1) a great instability of the vagosympathetic system, and (2) a local labyrinthine trouble.

"By experiments with pharmacodynamic substances (atropin, pilocarpin, adrenalin) Kobrak tries to find in each individual case whether it is a question of vagotomy or one of sympathicotony.

"The examination of these different theories shows the important place given to vasomotor troubles in the pathogeny of Ménière's disease."

The following history is recited in detail as being that of a typical severe case, observed over a sufficient period of time and in sufficient detail to satisfactorily exemplify the condition.

The patient, Mr. V. B., an executive, was first seen in April, 1924, and was then 49 years of age. He complained of intermittent tinnitus and vertigo for the preceding five years. Two exceptionally severe attacks had occurred two years and four weeks, respectively, before the first examination. Other slight attacks occurred at intervals of a few weeks. The first severe attack was preceded by increasing deafness in the right ear, associated with nausea and vomiting. The second severe attack was not preceded by deafness but the vomiting was protracted, lasting about one hour, after which the vertigo slowly disappeared.

At no time had there been any indiscretion of diet. The patient's habits were moderate; two cigars daily or less; alcohol only occasionally. Apart from an appendectomy in 1921, there was no severe illness. There was no history of ear infection, and nose and ears had been pronounced normal by a competent otologist. The teeth were in excellent condition; there was only one crowned tooth and one other devitalized tooth. The cervical glands and the thyroid were not enlarged.

The pulse, 76, was regular and moderate; vessel walls were palpable but not tortuous, and the temporal arteries were neither palpable nor visible. Blood pressure, 128/75. The physical examination was essentially negative. Urinalysis on various occasions was completely negative except once, in June, 1925, when there was a large amount of indican.

The following is an excerpt from the radiologist's report: "Emptying time of the stomach was normal, although the colon presents evidence of stasis and a moderately active degree of spasticity, especially along the descending colon and the sigmoid flexure. Pylorus somewhat spastic, but no evidence suggestive of gall bladder pathology. Kidney shadows clear and normal." Fractional gastric analysis was negative.

The patient first presented himself to the author during an interval. At this examination both membranæ tympani were neg-

ative, the nasal mucosa was slightly engorged but the septum was straight and there was no obstruction. The tonsils were moderate in size and apparently quiescent. There had been only one attack of acute tonsillitis, several years before. The nasopharynx was mildly congested, otherwise negative. The hearing tests with forks showed unimpaired function throughout the tone range on both sides, with the exception of a very slight loss of perception to C⁵ and above on the right side. No changes, either relative or absolute, were observed in the air and the bone conduction. There were no spontaneous vestibular symptoms; the station was good; there was no past pointing or nystagmus on looking in any direction. Turning and douching tests on several occasions were negative. These observations were verified by Drs. John B. Shapleigh and H. W. Lyman.

The attacks continued at intervals of a few weeks, usually while the patient was out of the city, so that no vestibular tests could be made during an attack until May, 1926, during a mild exacerbation. Rotary nystagmus to the right on looking right was observed, which lasted for an indefinite period; some horizontal nystagmus to the left occurred on looking left. There was a slight transient nystagmus to the left on looking up and down. Caloric and turning tests resulted in such violent discomfort as to necessitate discontinuing the tests.

"June, 1925. Attacks continue at intervals of several weeks, some with nausea, others without. Bowels inclined to move during attack.

"July, 1925. Attacks continue. Left first upper molar extracted. Some signs of pus about the root. The patient was put on a diet of buttermilk and bulgarian bacillus, which was followed by an increase of symptoms.

"From July 16th to 24th, 1925, dizziness daily. Toward the end of this period nausea and vomiting. Sometimes confined to bed."

Similar attacks at least once a month until May 4, 1926. On this date a nose and throat examination, which was otherwise negative, disclosed a pale, raised, sharply circumscribed patch of edema in the nasopharynx involving Rosenmüller's fossa and the

posterior lip and orifice of the right eustachian tube. While the wheal in this position was not suspected of causing the disturbance, it suggested the presence of allergy.

The allergist found the patient sensitive to milk, butter and cheese, which were eliminated from the diet.

From that date to the present writing, an interval of five years, the patient has suffered but three attacks, each following the ingestion of butter, once experimentally and twice accidentally. The patient is in excellent general health. Some perception deaf-

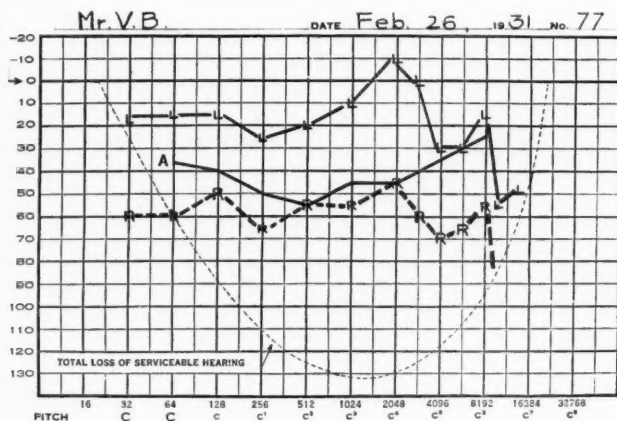


Fig. 1. Audiogram made between attacks. Those made shortly after a crisis showed only minor variations from this chart, which were confined to the left ear.

ness remains on both sides, which fluctuates slightly with his general status of health. The fluctuations, however, are more pronounced in the left or unaffected ear than in the right. Whether the right auditory nerve became permanently injured by the long persisting edema or not is a matter of conjecture, in view of the dental infection which persisted for some time.

The allergic analysis in this case is particularly instructive in that the patient is not skin-sensitive to the dairy products, although other positive reactions show him to be an allergic indi-

vidual. There was no history of hives, eczema or vasomotor disturbances. The mother at one time had eczema. The patient in childhood had an aversion to milk ("dislikes for foods are often important leads in children"), and the history was also suggestive in that attacks were worse while the patient was on a diet rich in milk and eggs. It was on the basis of this history that at his first visit milk and eggs were excluded from his diet. Beyond indicating the allergic background, skin tests were of little help. Reactions interpreted as mildly positive were those of horse dander, pyrethrum, pepper, oats, potato, corn, beans, beef, pecans and the pollen of ragweed; to all of which the patient may be exposed without disturbance.

There is an idiosyncrasy to iodides in that both potassium iodide and calcium iodide produce headache and increase the tinnitus.

Milk products ingested in the smallest doses were invariably followed by vertigo.

Cases such as this emphasize the need for a careful allergic history in addition to skin tests, as the latter are reliable in only 50 per cent of allergic patients.

The general examination and the allergic and radiologic investigations in this case were made by Doctors Walter Baumgarten, Charles H. Eyermann and Edwin C. Ernst, respectively, to whom I am indebted for their concise records.

The completeness of these observations, the length of time over which they were made, and the dramatic and immediate recovery of the patient following the elimination of milk, leave little doubt as to the correctness of the diagnosis. The three exacerbations, far apart in point of time and each one following the ingestion of butter, confirm it.

II. REACTIONS IN THE MIDDLE EAR.

More prevalent than one would imagine, unless one looked particularly for it, is an edema of the tympanum, including the membrana tympani, resulting from the ingestion of some foreign protein to which the patient is sensitive. The symptomatology is exactly that which might be expected of such an edema, in the absence of infection, upon the structures confined in the bony

tympanic walls. The outstanding signs are sudden severe pain, transient in character, that subsides spontaneously as does an angioneurotic edema elsewhere in the body; and the reduction of hearing attendant upon mechanical damping of the conduction apparatus. According to Lewis, who observed several cases, prompt spontaneous rupture of the membrana tympani with profuse serous discharge was the rule. This has not been the experience of the author, who has not seen a spontaneous rupture in a series of more than ten cases, although he recollects the experience of a surgeon who, several years ago, was subject to sudden spontaneous rupture with serous exudate which regularly healed without persisting perforation within twenty-four hours. This individual and his infant son have since been shown to be allergic individuals.

The history, as stated, is ordinarily that of intense pain, roaring and partial deafness of one or both ears, coming on suddenly after exposure to or ingestion of some foreign protein. The drum is red and swollen but has not the typical convexity of the bulging drum of infectious otitis media. Often the edematous epithelium gives it a whitish cast or bloom.

On incision, there is a sensation of cutting into a spongy structure and not a taut membrane. A drop of blood or of serum appears and the incision heals promptly without suppuration unless secondary infection intervenes. An afebrile course is the rule, but there may be an elevation of temperature as in any other anaphylactic reaction.

The diagnosis is made upon the appearance of the drumhead and upon the allergic history of the individual. The importance of this history, aside from the skin reactions, should be stressed, as the latter may be absent. Most of the author's cases have been in infants under five years of age, the more pronounced being in recently weaned infants who prove sensitive to some component of their new diets.

The following case is cited in detail as being typical. The patient, C. M., fifteen months old, was first seen in February, 1929, in consultation with a pediatrician who had then opened the ears seven times during the current attack.

The membrana tympani was a bloody mass, and there was a moderate amount of pus in the canal. The child was struggling with a severe attack of asthma at the time. There was a history of frequent asthma for the previous nine months, which adrenalin relieved promptly. The earache did not always accompany the asthmatic attacks, but asthma invariably accompanied the earache. The pediatrician felt that the recurrent ear infections precipitated the asthma attacks and promptly performed paracenteses.

After one of these, a spread from the ear showed numbers of cocci, a few leucocytes and epithelial cells. No eosinophils were observed. Spreads of the mucus from the nose made at the same time showed numbers of leucocytes, about 1 per cent of which were eosinophils.

At the next attacks the succession of events was carefully noted, and it developed that the earache did not precede the asthma but was rather coincident with it or followed it. On these occasions the drum membrane was not incised and the otitis media subsided with the asthmatic attack.

This child was skin sensitive to egg, potato, chicken feathers, cottonseed and horse dander (all four plus), and to wheat, goose feathers, duck feathers and cat hair (all two plus). On eliminating these contacts, the asthma improved to a striking degree and no recurrence of ear symptoms has been reported. The asthma has not entirely disappeared, but the mother explains that she has been giving the baby "a little" egg and potato.

In such cases as this one apparently nothing is to be gained by incision of the drumhead, as the condition subsides when the allergic crisis is relieved, and there is no infectious material to liberate. The repeated incisions which are prompted by the continuation of pain are sure to be followed by infection, and the resulting purulent otitis media continues after the cessation of the allergic attack. It is difficult to say where to draw the line in allergic individuals between allergic and infectious otitis media. The best indicators are probably the temperature, the general appearance of the patient, and the presence or absence of eosinophilia.

CONCLUSIONS.

Localized allergic reactions may simulate other ear diseases by the mechanical reaction of edema upon the tissues of the tympanum and the labyrinth.

They may be differentiated by the nature of the onset, often accompanied by allergic manifestations elsewhere, by a careful history of the patient's sensitivities, and sometimes by the occurrence of eosinophilia.

1010 BEAUMONT BUILDING.

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XIII.

EXTRACRANIAL ANEURYSM OF THE INTERNAL CAROTID: REPORT OF A CASE.

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Aneurysm of the internal carotid extracranially is not very common, and yet a sufficient number of cases is reported in the literature to demand care and caution in dealing with throat conditions. Up to August, 1925, Winslow¹ recorded 106 cases of extracranial aneurysm of the internal carotid, 42 of which were of the spontaneous variety.

Aneurysm of the internal carotid may be true or false; the true has a sac wall containing any or all of the arterial tunics, while the false has a sac wall composed of adventitious tissue. These aneurysms may be further classified as spontaneous, erosive or traumatic, depending on the causative factor. The spontaneous may be idiopathic, arteriosclerotic or syphilitic; the erosive may be due to absorption of the arterial walls, such as one may find in an abscess cavity, while the traumatic are most often found in gunshot or stab wounds.

The signs and symptoms of this condition are usually a feeling of fullness in the throat, accompanied by a throbbing sensation. On inspection of the pharynx, one usually sees a soft, elastic swelling with pulsation and usually a distinct bruit can be heard by placing a stethoscope over the mass; pulsation may, however, be absent or so weak as to escape detection. Externally there may be nothing visible or one may see a tumor behind the angle of the jaw. If the common carotid is compressed sufficiently to arrest circulation, both the pulsation and the bruit disappear as long as the pressure is kept up, and the swelling is reduced in size. Sometimes difficulty in swallowing is complained of as well

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as hoarseness, while there may be deviation of the tongue on protrusion.

Aneurysm must be diagnosed from sarcoma, syphilis, peritonsillar abscess, pulsating hemotoma, gland masses, etc.

Dubreuil² diagnosed an aneurysm as an adenoma or sarcoma and discovered his error on attempting to remove the mass. Winslow³ cites a case of his own on whom a physician had made a diagnosis of tonsillar abscess and made a stab wound, evacuating only blood; in the same paper he lists five cases diagnosed as abscess and which had been treated by puncture, and two cases which had been diagnosed as sarcoma. Shipley and Lynn⁴ quote Werner as having collected eleven cases of extracranial aneurysm with a correct diagnosis of only six, and says they are often mistaken for growths or abscess. Winslow¹ says Chassaignac lanced a retropharyngeal swelling for abscess, but fortunately cured the patient by ligation of the common carotid. This same author cites twelve cases of aneurysm of the internal carotid under twenty-one years of age, just the time when one frequently sees peritonsillar abscess. Therefore, one should always stop, feel and listen when dealing with swellings in the throat. The Wassermann will be helpful in eliminating syphilis. In peritonsillar abscess, the condition is acute and of short duration; there may be temperature and more or less severe pain. In a neoplastic mass there is not likely to be a bruit or pulsation.

In the treatment of this condition there is more or less unanimity of opinion that some form of ligation is necessary. In 1808 Astley Cooper successfully ligated the common carotid, thus curing an aneurysm, and in 1803 Fleming successfully performed the same operation for hemorrhage. In 1842, Syme tied off the common carotid for extracranial aneurysm of the internal carotid and in 1871 Briggs ligated the internal carotid above and below the aneurysm.

The operation of choice in this condition is ligation of the vessel between the sac and the trunk, and if this is not feasible, one should ligate the common and external carotid with all the branches of the latter on the cardiac side. Should the aneurysm return despite ligation, Mattas suggests extirpation or prefera-

bly obliterative endo-aneurismorrhaphy, provided the upper and lower poles of the sac can be secured for hemostosis.

The great difficulty in carotid ligation is the cerebral disturbances that may follow. Because of this Halstead has recommended temporary occlusion of the common carotid before attempting obliteration of the diseased vessel. Winslow³ says: "Although this precaution was not observed in my case, neither at the time of ligation nor subsequently did any harmful symptoms arise. Perhaps the lack of development of harmful symptoms in ligation for aneurysm is due to an already partially established collateral circulation."

The complications which may arise are hemiplegia, which, according to Johnson,⁵ occurs in 15 per cent of cases, or cerebral softening, which, according to Da Costa, is found in from 20 per cent to 25 per cent of cases; while Cryle says 50 per cent of those developing cerebral trouble die, yet he also says that the direct operative mortality is only 3 per cent. According to Horsley,⁶ the danger of ligating the common carotid increases enormously after forty years of age, and this is due to the lessened cerebral blood supply. He advocates as the best method of treatment extirpation with the double ligature.

Jordan⁷ says that in ligation of the common carotid 25 per cent develop brain disturbances, and 10 per cent fatal brain softening. He therefore recommends previous loose constriction of the carotid for 48 hours. His technic consists in a gradual occlusion of the main trunk until the peripheral pulse just ceases, in which case there is no damage done to the tunica intima and there is no clot formation. Johnson⁵ says that "the efficiency of the collateral circulation should always be tested preliminary to a ligation of this vessel." This may be done by temporarily stopping the circulation within the common carotid while watching the corresponding eyeground, as well as noting any dizziness or faintness complained of by the patient.

The prognosis after operation is very encouraging. Winslow¹ reports his case completely cured four years after ligation of the internal carotid, and Shipley and Lynn⁴ report a case symptomatically well ten years after operation. Since 1880, 59 cases have been operated on with 41 cures, 2 improved, one recurrence

and 15 deaths. Of the 30 cases not operated on, 20 died, 6 were unimproved and only 3 recovered. These figures surely demand operative interference.

REPORT OF A CASE.

Mrs. E. S. White, age 53, first seen April, 1930, complaining of swelling on the right side of the throat, which has been gradually getting larger for the past eighteen months. She discovered the swelling in her throat accidentally. At times she has felt a throbbing in the lump but never any pain.

The patient had usual children's diseases, one operation following childbirth and one miscarriage, followed by curettage. She has four boys and two girls, all living and well. Family history irrelevant.

Examination showed a patient about 50 years of age, rather thin and underweight, otherwise nothing of note externally. On looking into the throat one saw, filling the whole right tonsillar area, a rounded mass extending from the base of the tongue up to and under the soft palate. A distinct pulsation could be felt along the entire length of the lump, while a marked blowing sound could be heard with a stethoscope placed on the mass. Externally there was nothing to be seen, felt or heard.

Wassermann, repeated and provocative, were all negative, as was also the X-ray.

The pulse was 114, B. P. 184-94.

Patient was admitted to hospital for a more complete work-up and if possible a ligation of the right common carotid. It was then found that the blood sugar was 111 mgs. per 100 cc. of blood, with some glycosuria and a fairly marked increased metabolic rate, ranging between +77 and +49.

The consulting general surgeon, Dr. Paffert, agreed with the diagnosis of aneurysm of the right internal carotid and also the proposed ligation of the common carotid. It was felt that operative interference, however, was strongly contraindicated because of the blood pressure and the toxic goiter, and the consulting internists, Dr. Tasker Howard and Dr. G. H. Roberts, also strongly advised against any operation until the blood pressure was reduced and if possible the toxic goiter got under control.

With this end in view, the consulting internists put the patient on appropriate treatment, but after a day or two she left the hospital against all advice and we have not been able to locate her since.

CONCLUSIONS.

1. Aneurysm of the internal carotid is common enough to warrant great care in examining all throat swellings.

2. Ligation of the common carotid is the method of choice in the treatment.

3. The prognosis is good if ligation is done.

4. The mortality in untreated cases is high.

We wish to thank Dr. Paffert, Dr. Tasker Howard and Dr. G. H. Roberts for their kind cooperation in the general work-up of this case.

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XIV.

INTERPRETING SINUS ROENTGENOGRAMS.*

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I am going to take up very little of your time with this paper, so that we may have a liberal discussion of the reasons some of you derive much information from your sinus roentgenograms, some very little and some none at all.

There are many factors governing the making and the interpreting of sinus films, and I am sure there are some of you here who have not appreciated the difficulties in the path of the roentgenologist.

The most essential point in the interpreting of sinus roentgenograms is the quality of the films. It is absolutely necessary that they should be as nearly perfect as possible. Unfortunately, there are several factors entering into the technic of producing the films that make 100 per cent perfect films impossible at the first examination. Briefly, these are the bony thickness of the skull, the quality of the bone and the variation of the relationship of the sinuses.

These factors you are no doubt familiar with, but there is another condition which I do not think you consider. This is the personal preference of different surgeons for different densities of films. Some of you like a dense, extremely contrasty film, and some a thinner, less contrasty film. Both are entirely right because there are two classes of surgeons who see many films and who are familiar with interpretation, viz., those who consider the anatomy as giving all the information they desire, and those who prefer to see the minute changes in pathology.

In order to show the anatomy to the best advantage, it is necessary to use a dense film, which requires so much exposure and penetration that, in most instances, the finer shadows of the

*Presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society at Atlantic City, January 5, 1931.

soft tissues are burned through and only the gross pathology is shown.

On the other hand, a film of less density and less extreme contrast will show the minute bone changes in the ethmoid region, and the presence of finer changes in the soft tissue in the sinuses, but which necessitates more careful study to visualize the anatomic relationships. The films of this type are more valuable for proper interpretation but are less spectacular in appearance.

This personal preference is perfectly proper in the case of the surgeon who relies upon his own interpretation, but makes for complications if the films are subsequently submitted to a surgeon of the opposite class. The films are either too dense or too thin, and the roentgenologist who made them is blamed for poor work.

I should like to make a plea for standardized technic. If all films were made at the same angles, and using the same electrical factors, exposure and distance, we would obtain a set of films that would be appreciated by all surgeons. This can only be accomplished by deciding on a particular degree of density and angles that will bring out all the information visible on a film.

In this way, films made in different laboratories would correspond and the patient saved much time and expense. This also means that the roentgenologist and the surgeon must spend more time in studying the films.

The information you receive from a roentgen examination of the sinuses is directly proportional to the relationship of the three capital C's, viz., Co-operation, Consultation and Co-ordination.

Most of you depend on the roentgenologist's report, but he should be familiar with the surgical anatomy and the pathology expected. He should be a clinician, to a certain extent. Most roentgenologists are clinicians in a general way, but when it comes to this extremely special field he has to depend on your help. Here is where the three C's enter the picture. Pick your roentgenologist, and both of you go through a course of training in the other's field, to the extent of being able to speak each other's language.

From the above comments I do not want to be quoted as advising the surgeons to interpret their own films. I am absolutely opposed to such a procedure.

A sinus roentgenogram is not a microscopic slide or a chemical reagent. It is a maze of delicate lines that have a definite meaning, if you can translate them. Translation means study and comparison with the clinical evidence. It takes two to do it, and the lack of this combined study is what has retarded the advancement of sinus roentgenology.

How many of you study the films with the roentgenologist, and then ask him in to the operation, if one is done, or at least tell him what was found? Questionnaires are sent out with some reports asking for this information, but most of you are too busy to fill these out, or just neglect it. The roentgenologist is not always right, and by knowing his error he can educate his eye to learn why the error was made.

I am not going to discuss the meaning of the different degrees of density in the sinuses, due to gross pathology, except to mention one reason why the roentgenogram and the clinical evidence do not always coincide. A patient comes to your office and you find evidence of pus in one or more sinuses. He is sent to the roentgenologist, and you receive a report of clear sinuses. You are positive there was pus present at the time of your examination, so you chalk up a failure for the X-ray. Here is where you are overlooking a valuable piece of information. You probably applied adrenalin or cocain to the nose, and after a lengthy examination and manipulation, sent him to the roentgenologist. On the way the patient repeatedly blows his nose, and by the time the X-ray is made he has probably succeeded in draining the sinuses.

A case of this type makes possible the differential diagnosis between granulations and fluid. If your transillumination is positive, and inspection indicates pus, and your X-ray is negative, the indications favor the presence of pus and not granulations or polyp from a chronic process. Incidents of this kind occur most frequently in examination of the antra, and are the cause of much controversy, especially over the telephone. A note to the roentgenologist stating that pus was seen and adrenalin applied would simplify the wording of his report to you.

For the most easily appreciated outline of a chronic sinusitis, injection of a nonopaque fluid is the best method of procedure. There are certain difficulties of technic in occasional cases, but the resulting film is spectacular and satisfactory.

The extreme enthusiasm of this method has waned somewhat and is now settling down to a selection of suitable cases. It is an extremely valuable method of diagnosis, and, wisely and properly used, gives absolutely definite information of the condition of the walls of the sinuses.

A properly made roentgenogram, studied carefully with the stereoscope, will generally show the outline of dense granulations, but unfortunately many surgeons are not able to use the stereoscope and therefore miss these fine details. He has to depend on the roentgenologist for a graphic picture of the condition. An injection of an opaque fluid enables him to visualize this picture.

Now, what constitutes a properly made roentgenogram? I will not accept a set of films that do not show the cancellous structure of the frontal bone and parietal bones. A film that shows this detail clearly will show the finer detailed structure of the sinus walls. I like a density and contrast that will visualize the ethmoid septa. Too much contrast will block out the detail. The lateral views must be made stereo, and preferably also the vertex mental view. There should be an anteroposterior view at the 23 degree angle and a Waters position. These four views are made routinely, and after careful study others, at different angles, can be made, if necessary, to clear up a doubtful point. In the majority of cases, these four views are all that are needed.

Most interpretations are based on the obliteration of the sinus cavities, but I would like you to consider a change which occurs in the walls of the ethmoid septa, even in cases which do not show change in the contents of the cells. Stereoscopic lateral films are necessary to detect these changes, and they must be carefully studied. These septa undergo marked changes and indicate pathology when the anteroposterior view shows clear ethmoids. In a normal case the outline of the ethmoid labyrinth should be clearly seen, and every cell wall sharp and clear. An acute or early chronic case will show fuzzy semitransparent cell walls,

with possibly some of them missing. This appearance, as revealed by the microscope, is due to a phlebitis, and is termed on our reports as "an edema of the walls." The older chronic cases show a thinning of the walls, but clear cut outlines as though they had been compressed by a roller. This is due to a condensing osteitis. These conditions may involve the entire ethmoid labyrinth or simply one cell. Hence, you can see the absolute necessity of securing perfect detail in the film.

As I will show you on the slides, you can have a perfectly normal appearing ethmoid region, shown on an anteroposterior view, and an entire absence of bony septa on the lateral view, even in the absence of any surgical procedure. We have cases of low grade chronic sinusitis which show extremely fuzzy, edematous cell walls, some of them so transparent they are almost invisible. After a course of several months' treatment, a second X-ray examination showed a marked change in the septa. They had become more clearly visible. The fuzz had nearly disappeared, and those which were nearly transparent had assumed a more normal appearance.

There is also the opposite condition, an osteitic thickening, which is shown by an increase in the density and thickness of the walls. Cases of this type are naturally more easily distinguished and require less study to interpret them. This appearance is seen in the frank chronic sinusitis which can be seen on examination of the nasal cavity.

The changes, illustrated by the slides which we will see, have been very carefully checked by the microscope. The cases showing a change after treatment are from a series of examinations, for progressive deafness in children, made by Dr. E. P. Fowler and myself and presented before this society in 1929. These changes are shown in all of the cases of eye involvement which have been improved after operation on the ethmoids, and in most of which there was no apparent change in the appearance of the nasal mucosa and no other evidence of sinus infection.

I am asking you, therefore, in your study of roentgenograms, to observe more closely the bony structure of the ethmoid cells, and by the use of the three C's help us all to learn the reason

for these bony changes in the absence of macroscopic changes in the nose.

In conclusion, I should like to suggest a method of reporting cases which we have found very satisfactory at the Manhattan Eye, Ear and Throat Hospital. Reports coming from different roentgenologists vary so much in terminology that it is difficult to appreciate the degree of trouble they are referring to.

We have divided the degree of involvement into four divisions:

+ is the slightest change which can be detected.

++++ is the extreme degree of involvement.

+++ and ++ are subdivisions which will adequately express the degrees between these extremes.

This method is simple to dictate, simple to read, and forms a basis for comparisons at subsequent examinations.

XV.

GRADENIGO SYNDROME: AN UNUSUAL CASE.

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In 1904, when Gradenigo¹ drew attention to the symptom complex which now bears his name, he stated that he had previously, during that year, reported six cases before the Royal Academy of Turin, with the result that his colleagues, Mongardi, Ricci and Citelli, had turned over to him the histories of three other cases. In the same year Lannois² and Ferran,² Lubet-Barbon,³ and M. Cheval⁴ described four cases in which there was paralysis of the external rectus of the eye and temporoparietal pain associated with otitis media. In 1907, Gradenigo⁵ had collected and tabulated 65 cases (57 previously reported and eight of his own), many of which were discussed in detail in his report.

In 1910, Perkins⁶ reported 95 cases illustrating the syndrome, and, in his study of these cases, made many valuable observations. At that time he expressed the opinion that abducens paralysis per se was not an indication for operation, but that the severity of the temporoparietal pain was to be considered. In a later paper¹⁴ on the same subject, he said: "While recovery from the Gradenigo syndrome is possible without operative intervention, I believe that the syndrome itself, without apparent mastoid involvement, is sufficient justification for a mastoid operation, at least."

In 1926 Sears,⁷ in a most comprehensive report, collected all the cases reported in the literature up to that time, and added to this number 27 cases previously unpublished, except one which he himself had reported fifteen years before. In that report he tabulated findings in 172 cases, wrote a detailed study of the anatomy of the petrous tip, and summarized the observations and opinions of many other writers as well as his own.

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Since 1926 there have been a few case reports⁸ in the literature, and there have undoubtedly been many cases of this type which have been seen and not reported.

Textbooks, with a few possible exceptions (notably Perkins⁹), give very meager accounts of this syndrome, and are of very little aid to one seeking instruction as to the management of such cases, and little information as to prognosis.

There have been many opinions regarding the cause of the phenomenon. These range from the so-called reflex theory of Moos and Urbantschitsch¹⁰ to that of Goris,¹¹ who found a mobile sequestrum at the petrous tip which he removed surgically. Some cases undoubtedly were caused by sinus thrombosis, the involvement centering chiefly in the petrosal sinuses and in the cavernous sinus. Perkins says: "It is very probable that simple lateral sinus thrombosis could not produce abducens paralysis, per se, but only when the thrombus has extended to the superior or inferior petrosal or cavernous sinuses." In this regard, Wheeler¹² states that the abducens actually enters the cavernous sinus and passes forward through the blood of the sinus, this intimacy of the abducens with the blood stream accounting for the especial vulnerability of the nerve to sinus infection, toxemias from many forms of infection, and the many cases of abducens paralysis following spinal anesthesia.

The rôle of meningitis, labyrinth disease, abscess in posterior and middle fossæ, and osteitis of the petrous tip, were pointed out in Perkin's⁶ paper. Osteitis of the petrous tip and abscess in the posterior fossa were apparently the most frequent causes of the syndrome. Gradenigo assumed it to be caused by a circumscribed purulent or simple serous meningitis localized toward the tip of the petrous bone and determined by the diffusion of infection in the body of the mastoid.

In the case reports of the many observers of the Gradenigo syndrome I find no mention of any major complication except a rapid spread of the meningitis, which inevitably caused death. A large majority of the cases cleared up with no evidence of residual paralyses. However, Gradenigo⁵ noted in nine of his cases evidence of meningeal irritation—e. g., photophobia, nocturnal encephalic cries, spasm of orbicularis palpebrarum, fever,

vertigo and vomiting, rigidity of the neck and spasm or paresis of the facial nerve. In discussing the facial paresis which occurred in two of his cases, Gradenigo³ explained that this involvement probably came about as a result of the inflammation of the facial nerve in its course through the tympanum. He did not feel there was any localized central lesion about the nuclei of the abducens nerve and the facial nerve, as suggested by Negro in the discussion following the presentation of his cases, nor did he mention the possibility of its being the result of localized meningitis. No mention was made of symptoms or signs indicative of a more or less benign extension of the pathologic process.

Assuming that Gradenigo's syndrome is frequently caused by a localized meningitis, either purulent or simple serous, is it not logical that a simple serous type of meningitis might spread as such to other areas, involvement of which might produce symptoms in distant parts of the body? And, since the venous pathways of the dura are simply channels surrounded by dura, is it not possible that a serous meningitis might have its origin from these channels, producing a localized or patchy inflammatory reaction in the meninges in the vicinity of the venous sinuses?

In the following case report these assumptions were made and the treatment instituted was largely based on them.

REPORT OF CASE.

W. C., a boy, age eight years, had, when two years old, a tonsillectomy followed by considerable scarring of the soft palate and of the tonsil pillars. At the age of seven he had whooping cough complicated by bronchopneumonia. On March 2, 1930, he again had bronchopneumonia, which lasted until March 30, 1930. On March 22nd he developed an acute purulent otitis media of the right ear, prior to which his temperature ranged from 100 to 103.8 degrees F., with daily remissions. At the time myringotomy was done his temperature had risen to 105.2° F. Only a small amount of thick, sticky, yellowish pus came from the incision made in the ear drum, and unfortunately no culture was made. Within a few hours, however, the temperature began to fall, coming down by lysis, until normal temperature was reached on March 30th. On that day he was allowed to leave the hospital in the custody of his parents.

On April 9th, he was readmitted to the hospital with a temperature of 101.2° F., suffering from intense temporoparietal pain, and complaining of double vision, which had appeared three days previous to readmission. There had been no discharge from the right ear since leaving the hospital, nor had there been any pain in or behind the ear. His general condition was very poor, as shown by pallor and extreme loss of weight. Leucocyte count 14,300 with 77 per cent polymorphonuclear cells. On examination the right ear was dry, the membrane tympani normal in color, thickened, lusterless and landmarks indistinct. There was no drooping of the posterior canal wall, nor was there any mastoid tenderness except possibly some slight discomfort on pressure over the mastoid antrum region. There was paralysis of the external rectus muscle of the right eye, and some facial weakness of the right side of the face, manifesting itself chiefly by immobility of the risorius muscle. There was no stiffness of the neck, abdominal reflexes active and equal, knee jerks active and equal, plantar reflexes indecisive, and there was a suggestion of ankle clonus on both sides. An X-ray of the right mastoid showed definite clouding of the mastoid cells.

The same day, April 9th, at 9:30 p. m., a right simple mastoidectomy was done. The cortex was thick, the mastoid process being of the diploic type, the cortical cells of which showed a mild congestion, with no granulation or pus. The cells about the aditus, however, contained a small amount of granulation with a sticky mucus exudate, and there was some breaking down of cell septa. The dura and sinus were normal in appearance. A culture taken from the aditus region showed the presence of streptococcus—not mucosus capsulatus.

April 16th, the eye movements of the right eye were nearly normal, and the facial weakness was barely noticeable. However, during this period his temperature ranged from 100 to 104.6 degrees F. The blood culture was negative.

April 21st, at 11 a. m., the patient complained of headache and a prickling sensation in the left hand. At 1 p. m. there was an incomplete spastic paralysis of the left upper extremity followed in a short time by the same condition in the left lower extremity. Accompanying these paralyzes there was weakness of the left

side of the face. At first the patient was able to raise the arm with great effort and to flex the elbow, but he was unable to pronate or supinate, flex or extend the wrist or move the fingers, which were held tightly in flexion. Later mobility of the left arm was almost completely lost. Deep reflexes of the left arm and leg were hyperactive, while the abdominal and cremasteric reflexes were absent. Babinski positive. Marked muscular weakness and lack of co-ordination in the left lower extremity. Drooling from the left side of the mouth.

April 22nd. A transfusion of 250 cc. citrated blood was given.

April 23rd. Patient was irrational at times and had a typical nocturnal encephalic cry. There was no change in the condition of the left upper extremity. Abdominal and cremasteric reflexes were returning, however, and in the left lower extremity there was some increase in muscular power, but movement was purposeless and lacked co-ordination. Babinski positive. Examination of fundi oculi showed merely a slight venous congestion. Lumbar puncture was done and a clear fluid under little if any pressure was obtained. Laboratory examination of the fluid disclosed only seven cells, with no chemical changes.

April 25th. A second transfusion of 260 cc. direct from donor to recipient was given. The only apparent reaction noticed was a pronounced involuntary twitching of the lower part of the face, which lasted only a few moments after the transfusion. The patient could now show his teeth, whistle, close his eyes tightly and wrinkle both sides of the face with ease. There was some extension of the fingers of the left hand, some pronation and supination of the left forearm. There was more power and less inco-ordination of the left lower extremity as well as the upper. Finger-nose and heel-knee tests were fairly well done. Babinski still present on the left side. Examination of eyegrounds revealed practically normal fundi.

April 26th. The child was brighter, took an active interest in his surroundings and played with his toys, using both hands. His reflexes were normal and equal.

April 30th. Child was discharged from hospital. He had been out of bed for the past two days.

This child was seen one month after discharge from the hospital, and there were no residuals. He had gained weight, had good hearing and appeared to be in excellent health. His father has recently reported to me that he is perfectly well.

SUMMARY.

In this case the first symptoms were those of the Gradenigo syndrome, plus a facial paresis on the same side, which practically cleared up seven days after simple mastoidectomy, to be followed five days later by spastic hemiplegia of the opposite side of the body, coming on gradually, first involving the arm and then the leg, and accompanied by photophobia and encephalic cries. There was no stiffness of the neck, only a slight venous congestion in the eyegrounds, and no increase in pressure or change in constituents of the cerebrospinal fluid.

In addition to being a delicate child, the boy had recently had pneumonia, which was slow in resolving and from which he had almost succumbed. The original acute otitis media which had arisen while he was still running temperature with the pulmonic disease, had subsided and the ear had been dry for ten days prior to the appearance of Gradenigo's syndrome.

A thorough simple mastoidectomy was done. In spite of the very meager pathologic changes found, the pain and ocular palsy began to improve thirty-six hours after the operation, and within a week had practically disappeared.

The subsequent appearance of a contralateral spastic hemiplegia was a recrudescence of the former serous meningitis, which now involved the motor area in the middle fossa.

On the assumption that the symptoms and signs were caused by localized serous meningitis, which probably resulted from infection of the petrosal sinuses, transfusions were given with very striking results. Whether the benefit obtained resulted from an improvement in the general condition of the patient or from the retardation of the local process, the writer is unable to state.

COMMENT.

It is possible for a localized serous meningitis to clear up in one locality and later manifest itself in another, causing an entirely different train of symptoms.

In view of the fact that many cases¹³ exhibiting the Gradenigo syndrome are individuals in whom, for some reason or other, the natural resistance has been lowered and in some of whom there is a possibility of infection or irritation of the petrosal sinuses, a valuable aid in the treatment of these cases after a mastoid operation has been done, is repeated small transfusions.

As far as the writer is aware, no case reports similar to this one have thus far appeared in the literature.

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XVI.

THE VALUE OF THE SCHILLING HEMOGRAM IN THE OTOLOGIC INFECTIONS.

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Determination of the total leucocytes and the differential count are very common laboratory procedures and are generally regarded as two of the simpler blood analyses. Until comparatively recent times, the Ehrlich classification of the various cellular elements has been employed in routine examinations, as the more elaborate observations introduced by Arneth in 1904 were so exacting as to prevent their universal adoption. The Arneth count, however, found considerable favor among several of the contemporary hematologists, and gradually the method became better understood and appreciated. Thus, its subsequent modifications by Cooke, and more recently by Schilling of Berlin, have been well received by those who previously had substantiated and verified the practical value of the "nuclear deviation" of the leucocytes in various pathologic states. A further valuation of the hemogram, as related to various infections, particularly mastoiditis, is presented through the study of nine such cases.

THE DEVELOPMENT OF THE HEMOGRAM.

Ehrlich early demonstrated that there were intermediary types among the polymorphonuclear cells, and Arneth, about 1902, observed that in patients suffering from infectious diseases these cells were found much more frequently than in normal controls. Further study led to his classification of the neutrophiles grouped according to the number of their nuclear segments. He assumed that the neutrophilic cells having one or two nuclei were relatively immature, less resistant and less able to combat infection than the older ones with more nuclei. All the polymorphonuclear cells were found to fall into one of five large classes according to the number of their nuclei. These numbers were placed in a

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horizontal row, beginning on the left with Class I—cells containing one nucleus, and ending on the right with Class V, containing five nuclear segments. From a count of fifteen normal persons a normal neutrophilic blood picture was constructed:

I	II	III	IV	V
5%	35%	41%	17%	2%

An increase of the younger cells and a decrease of the more mature elements, with the ratio more marked in proportion to the severity of the infectious process was designated a "drift," or displacement to the left. As the infection subsided the nuclear deviation again shifted to the right and back to the normal percentages.

Owing to the intricate subclassifications, the Arneht differential required considerable experience and time for its enumeration. Various cell types were classified differently by individual observers, leading to inconstant interpretations. Cooke, in 1908, further elaborated the method through his studies on the degree of nuclear complexity of segmented forms, by which he demonstrated that a finer degree of right deviation might be ascertained. V. Schilling first reported the value of hemogram studies in 1912, his observations being restricted to tropical disease manifestations. Later, his researches carried him into practically every disease process, and he came to view the normal blood picture as a complex of highly differentiated cells in various stages of maturity and in very constant proportion, the peripheral elements representing a part of a biologic chain of events mirroring the functional status of the hematopoietic tissues. Furthermore, Schilling introduced the triadistic conception of the origin of the cellular blood constituents, namely, that the erythrocytes, platelets and granulocytes have the bone marrow as their primary source and site of formation, while the lymphocytic series are derived from the various lymphoid tissues, and the monocytes (large mononuclears and transitionals) are believed to be representative of the reticuloendothelial system described by Aschoff and Kiyono in 1913.

Under normal conditions, the three white blood cell groups mature in their respective mother tissues, and a quite constant number are released into the blood stream, where the remaining

life cycle is run until the old cells are withdrawn and replaced by fresher elements. Despite the many physiologic factors present which tend to be manifested by changes occurring in the blood stream, such as exertion, menstrual, digestive and temporal variations in the white cells, the relative quantitative and qualitative features are maintained at a remarkably consistent level. In disease states, however, the normal physiologic mechanisms are no longer in force. There is present an irritative factor (or factors)—alien elements introduced into the body—generally considered and included under the name "toxin," whose character determines the resultant type of reactive response of the sensitive hematopoietic tissues. Weiss has pointed out that if the toxin is stimulating in nature, a hyperleucocytic response of a regenerative variety results; if inhibitory in type, the hematopoietic centers react with a leukopenic response with no evidence of juvenile forms; while if both stimulating and inhibitory factors are present a regenerative-degenerative response is noted in the blood stream. The hematopoietic organs are altered and the degree of stimulation, inhibition or combination of these elements to which the bone marrow, lymphoid tissue or reticuloendothelial cells are individually and collectively subjected is reflected in the peripheral blood stream.

The irritation can, and usually does, involve one of the three hematopoietic sources more than the others. The toxin lowers the barriers to the circulation, with the resultant liberation from the parent tissues of more cells, which become progressively less mature as the stimulating agent increases in intensity. Schilling devised a scheme whereby all the various degrees of reaction occurring as the result of functional imbalance of blood tissue might be viewed in graphic perspective. Using the older Arneth horizontal scheme, he divided the cellular blood components and recorded them as follows:

Date	Red blood cells	Hemo- globin	White blood cells	Baso- phils	Eosino- phils
Myelo- cytes	Young cells	Staff cells	Segment nuclei	Lympho- cytes	Mono- cytes

It is evident that the principal new feature of the Schilling hemogram is a less involved differential of the granulocytes than was attempted by Arneth and yet a more comprehensive division of these elements than was possible by the Ehrlich classification.

A full description of the various morphologic characteristics of the blood cells is unnecessary, but the newer neutrophilic and monocytic types, as described by Schilling, must be carefully visualized in order to appreciate the full significance of his contribution:

The myelocyte is of course the very immature granulocyte, and is never present in the normal blood picture. The neutrophilic variety is somewhat larger than the mature neutrophilic leucocyte. The cytoplasm takes a pale blue color with polychrome stains, and the granules are usually fine, delicate and variable in staining reaction. However, at times, the granules may be quite coarse, irregularly developed, and deeply staining. The nucleus is round, oval or kidney shaped, faintly staining, distinctly trabeculated and vesiculated with coarse granules and usually contains nucleoli.

The juvenile neutrophilic leucocyte (Jugendliche; metamyelocyte of Pappenheim) is rarely seen in the normal blood stream. These cells are more mature than the myelocyte, but like them are somewhat larger than the mature neutrophile. The nucleus is sausage shaped or beanlike, with a distinct indentation or notch, vesicular, moderately staining, and contains scattered "circumscribed chromatic nucleoli in the end bulbs." The cytoplasm is somewhat blue and contains moderate sized granules which usually are not so distinct. These cells are seen particularly in the severe regenerative types of infection.

The Stabkernige or Staff forms, generally grouped as "stabs" in this country, are the type which Schilling stresses as being very important. He considers them as neutrophiles which, due to degenerative inhibition of development, mature "without the partition of the originally sausage-shaped nucleus into segments." The "rod nuclear" forms have all the characteristics of mature neutrophilic cells with the exception that the nucleus is ribbon formed, of the same thickness throughout, and occurs in V, U, T, Y, S and E configurations. The degenerative stab forms are recognized by the narrow bands, twisted and often bizarre, and

the intensely staining, pyknotic structureless vacuolated nucleus. The cytoplasm contains highly stained and at times partly dissolved granules. These forms are quite fragile, hence are often observed to be distorted in ordinary smear preparations.

The "segmented nuclear" forms make up the majority of the neutrophils in the normal blood picture. The cytoplasm is typically white or pale pink, containing evenly distributed, fine, regular, neutrophilic or azurophilic granules. The nucleus usually has two to five irregular segments which are connected by fine threadlike bridges.

Cooke clearly indicated the character by which a lobe could be recognized—"if there is any band of nuclear material except a chromatin filament connecting the different parts of a nucleus, for the purpose of the count, it cannot be said to be divided."

All the lymphocytic cell series are included in one grouping, regardless of size. Schilling, likewise, placed under the heading "monocyte" the transitional and large mononuclear cells. These types are generally larger than the granulocytes. The cytoplasm is smoky blue to pale violet, showing very fine pink "dust" and small vacuoles, inclosures or plasmosomes. The nucleus is fairly large, oval or bean-shaped, usually slightly eccentric, with some tendency to polymorphism (lobulation). The structure is delicate and indistinct, and nucleoli are occasionally seen.

In summary, the various percentages of the several cell types comprising the normal hemogram are within the following limits:

R. B. C.	Hb.	W. B. C.	B.	E.	My.
4,500,000	90	6,000	.5	3	0
to	to	to			
5,000,000	95	8,000	(0-1)	(2-4)	

Juv.	Stab.	Seg.	Lymph.	Mono.
0.5	4	67	23	6
(0-1)	(3-5)	(54-72)	(21-35)	(4-8)

INTERPRETATION OF THE HEMOGRAM.

The clinical aspect and interpretation of the hemogram can only be discussed in its broader relations to acute infectious processes. Introduction of a foreign substance of toxic or infectious char-

acter results in the immediate stimulation of the body cells, both locally and generally. The bone marrow, generator of the combatant neutrophilic cells, responds quickly and specifically, and to a degree generally demonstrable by the increased total leucocytes and granulocytes. Such a cell response to an invading pathogen is largely determined by the virulence of the latter and the extent of the resistive powers of the individual. The blood picture, properly visualized, offers a means of further and better evaluation of the patient's resistive status. It is through this means that it is becoming possible to determine grossly whether the opposing forces of the host have completely checked, temporarily arrested or succumbed to the invasive agent.

It has long been a rather general conception that the total white cell count serves as an index of the patient's resistance—within certain limits, the higher the numerical response the more satisfactory the patient's reaction to his infection. Likewise, many have regarded the total polymorphonuclear percentage as a gauge of the relative severity or intensity of the infection. Weiss² points out that such a tenet frequently caused observers to fail to understand why, clinically, with a cell count of 6,000, a ruptured appendix was found at operation or a pneumonia with 9,000 rather than 25,000 cells. He further states that "at present the above conclusions have given place to the much greater information available by careful morphologic examination of the neutrophils, leucocytes and monocytes, and which now are assuming definite value in diagnosis, prognosis and treatment."

The total "stab" cell percentage is considered by Schilling and other observers as being far more valuable clinically or practically than the determination of the total white cells or the neutrophile proportion. These cell types have been definitely shown to be an index of the severity of the infection, or, in other words, a quantitative measure of the patient's resistance. Such a concept can be well understood when it is realized that these "stabs" are intermediary cell forms—between the quite immature myelocyte on the one hand and the mature polymorphonuclear or segmented type on the other. As they bear the brunt of the infectious activities, their numbers fluctuate proportionately.

The eosinophiles are the most responsive of all the blood cells, as they react more specifically and quickly than even the neutrophils. At the onset of the infection they definitely decrease and totally disappear during the course of every severe infection, but again quickly reappear as the condition subsides, and for a time may be present in considerably elevated number. The eosinophiles and monocytes are apparently closely related, as both quickly diminish or completely disappear during the early invasive or combat stage and return together in the monocytic or conquest stage. The lymphocytes decrease generally at the expense of the neutrophilic increase, so that during the severe stages of the infection there is usually a neutrophilic leucocytosis with an associated lymphopenia. If the latter persists and is marked it is considered a bad prognostic sign. The healing stage is characterized by the steady increase in the lymphocytes and during convalescence there may be a moderate elevation of these cell types above normal. From such facts, Schilling conceived the chain of biologic events that occur in the course of an infection to consist of the following:

1. The neutrophilic, resistive or combat stage, characterized by neutrophilia, shift to the left, lymphopenia and monocytopenia. This initial stage is followed (in favorable reactions) by:
2. The monocytic, defensive or conquest phase, characterized by return of the eosinophiles, increased monocytes, reduction of neutrophiles, yet continued shift to the left. Following the crisis of the infection the final third stage appears:
3. The lymphocytic, healing or recovery phase, characterized by the shifting back to normal, lymphocytosis, eosinophilia, decreased neutrophilia and monocytes.

The interpretation of the hemogram requires much experience, and existing biologic conditions, such as allergy, individual responses and peculiarities, age and other factors, must necessarily be evaluated when present along with the better known pathologic processes seen in infections. Schilling,¹ Weiss,^{2,3} Howe,⁴ Piney,⁵ Niehaus⁶ and others, while realizing that the clinical interpretation of the hemogram is primarily an individual problem, still drew several conclusions. The more general and con-

sistent of these, particularly as related to prognosis, may be summarized as follows:

1. The greater the stimulation of the bone marrow, the less mature are the neutrophilic cells thrown into the circulation, so that slight irritation produces an increased response in the normal segmented number, with stronger stimulation, staff and then juvenile forms appear, and in the very severe cases myelocytes may be found in the peripheral blood stream.

2. The total leucocyte count, stab cells and temperature do not necessarily concomitantly elevate. Usually with the height of the infection there is a corresponding peak in the stab cells, the latter dropping as the infection is overcome. In severe cases a sudden fall in the total count may be interpreted as an omen of good recovery, whereas the presence of many immature forms in the hemogram indicates the reverse.

3. Slight or definite leucocytosis with moderate nuclear shift (not more than 4 per cent juveniles and 12 per cent band forms) and decrease of eosinophiles and slight lymphopenia comprise a picture from which no clear prognostic inference can be drawn. It indicates an infection, however, and the need of further examinations at short intervals.

4. High leucocytosis with great shift to the left, with great decrease or even disappearance of eosinophiles and lymphopenia, points to a grave condition, particularly when there is a preponderance of young cells over the band forms.

5. Persistent progressive fall of the total white count with an extreme and increasing lymphocytopenia occurs with associated progressive decrease of total leucocytes and nuclear shift to the left.

6. The reappearance of eosinophiles is the earliest favorable sign when associated with increasing monocytes and lymphocytes. The basophiles are always found in increased numbers in severe infections when the defense factors are losing ground and tend to disappear with decreased function of the bone marrow.

7. It is the *continued high level* or the steady increase in stab cells without possibility of removing the causative focus that almost always spells a fatal prognosis. With removal of the purulent focus, the stabs will invariably drop, rapidly or slowly, de-

pending upon the amount of reactive or infective tissue left behind. A charted stab count coincides often more accurately with the course of the illness than the temperature (Weiss).

8. The hemogram also gives warning of various complications, such as further extension, sepsis, hemorrhage, etc., as the blood reacts more quickly than the temperature.

9. "It is not the blood that deceives" (Schilling)—i. e., findings are not always easily interpreted.

10. "The blood picture must always be observed together with complete clinical findings. Definite and certain clinical symptoms must never be disregarded because of a negative blood picture, nor should marked blood findings be disregarded because of absence of clinical symptoms."

CASE REPORTS.

This investigation was undertaken primarily to evaluate the hemogram as a possible diagnostic and prognostic aid in cases of acute otitis media and mastoiditis. The question of when operative interference in these cases is indicated is still discussed in open forum, and "such correct selection of the optimum time and appropriate case is a matter of delicate judgment and any rational additional aid is welcome." As the work progressed, other more specific features were felt worthy of careful consideration and may be briefly enumerated as follows:

1. The relation of the total white blood cell count and polymorphonuclear percentage, pulse and temperature recordings to the number of "stab cells."

2. Comparison of adult and juvenile hematopoietic response.

3. Factors of individual resistance and other variations.

4. Correlation of hemogram, clinical, operative or postmortem findings.

A careful technical procedure when once established was not altered throughout the series. Daily or quite frequent blood studies were taken at as near as possible the same time of each day in order to establish controlled estimation of the various physiologic states which are known to influence the blood picture. This element of time is considered by Kopetsky and Weiss as quite essential in hemogram interpretation. The total red,

white cell and hemoglobin determinations were carried out with standardized pipettes, counting chamber and color chart. Smears were given special attention in their preparation. New, fat-free glass slides were employed and two acceptable smears were made—criteria of acceptability being even, uniform, moderately thin and nonmarginated preparations. It was found that undue pressure or too hasty spreading of the blood over the slide produced an increased distortion of the quite fragile, less resistant, immature neutrophile cells. Likewise, examination of rapidly dried (heated or air blast) blood smears often showed a large but not uniform percentage of the different types of white cells destroyed. For these reasons the blood drop was lightly and rather slowly smeared and allowed to dry without attempt to hasten the process. This required about five minutes. Wright's stain was found entirely satisfactory as a means of cell differentiation if all the technical details of this procedure were carefully observed. The wet, stained slides were likewise allowed to dry without acceleration.

Counting was materially aided by the use of a mechanical stage. It was noted that the smears in which margination of the leucocytes occurred showed a distinct tendency of the immature neutrophilic forms (stabs, particularly) to collect on the edges in relatively large numbers. If such areas were to be taken alone as fields for estimation of the various cell percentages, a high total polymorphonuclear count, and especially an unduly elevated percentage of "stabs" and other immature forms, would be recorded and interpreted. The lymphocytes are less prone to be marginated, and due to their prognostic importance it follows that methods of counting the cells should include central as well as the more peripheral fields. With carefully prepared smears it was observed that undue margination could for the most part be avoided, and by the use of the "four field meander," or similar method of systematic examination of the slide, a fairly uniform differential was obtained. The personal factor in counting and differentiating the white cells deserves considerable stress. Just as one hemogram determination and interpretation is quite unreliable, so likewise it is undesirable for various observers to make infrequent hemogram studies on the same patient. Fur-

thermore, if the observer is in daily touch with the patient's clinical course, the latter can unconsciously affect his hemograms. For example, if the patient takes a sudden clinical turn for the worse, the classification of a questionable neutrophile is determined more by the symptomatology than by morphology. To eliminate this factor the patient's smears were labeled and dated at the bedside and then were stained along with others. A slide was then studied and classified without attention to its identity until the percentage figures were duly recorded. As a further check, 100 cells on the second slide were also counted and the average percentages computed from 200 cells. This procedure was particularly carried out during the first few observations in order to establish an accurate "pathologic norm" of the patient's blood cells.

A brief summary of the methods employed would include:

1. Daily blood studies taken at the same hour and examined by the same observer throughout the series.
2. Use of carefully prepared, labeled and stained smears, in which 100 cells were counted, unbiasedly, on two slides with calculation of average cell percentages.

Graphic representation of the serial hemograms was found to be of distinct clinical value. The temperature, pulse, total white cells and total stab cells were charted—the latter cells being looked upon as a daily manifestation of the patient's resistive reaction to the infection. The following case studies are not picked, illustrative ones, but are presented in the same numerical order as they were observed, and it is believed consequently offer a fairer means of evaluation of the hemogram in the diseases studied:

Case 1.—C. G., white female, age 65, came to Barnes Dispensary February 26, 1930, on account of pain, discharge and deafness of the right ear. The condition began three weeks previous with an acute nasal cold and pain in ear for four days. At that time the drum ruptured spontaneously and has since continued to discharge. For the first week following the rupture of the drum the patient was without pain, but in the last few days she has been conscious of a continuous dull, throbbing pain in this ear.

Examination revealed a patient who looked ill. The right ear canal was filled with a profuse mucopurulent discharge which came from a posterior superior perforation. There was also a slight sag of the posterior superior canal wall. The discharge was pulsating. The entire mastoid area was tender to touch. The nose showed the residuals of an acute pansinusitis. The tonsils were small and showed evidence of chronic infection. The patient was sent into the hospital for further treatment and operation. The examination there was as above with the addition that the urine was negative except for a trace of albumin, the Wassermann was negative, and culture from the ear discharge showed a hemolytic streptococcus. Mastoid X-rays, February 26, 1930, revealed a complete clouding of the mastoid structure of the affected side as compared with the other side, which was a normal pneumatic mastoid.

Further progress of the case is charted under hemogram studies.

Comment.—In this case it is noted that the total white cell count mounted rapidly but the stab cell and the polymorphonuclear percentage followed slowly. The temperature never was elevated over 38° C. and was quite normal the day of operation, but the latter disclosed considerable pathology, especially in the antrum. After postauricular drainage was established the stab cells promptly returned to normal limits and stayed so despite the fact that the external canal and operative site drained for over three weeks. This indicated that no serious postoperative infection was present and was borne out by the subsequent clinical course. The age factor, no doubt, influenced the rate of healing.

HEMOGRAM STUDIES—CASE I.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B. M.V.	Mt.	Std.	Seg.	Lvs.	Mn.	REMARKS
3-26 4:00 P. M.	37.2	85	4,090,000	80	10,700	1	1	1	8	64	22	3	Slight shift to left of hemogram. Paracentesis of right M. T. Much increase of serous discharge apparently under pressure.
3-1 9:00 A. M.	37	80			12,850	3			72				Discharge in right ear profuse and mucopurulent. Some mastoid tenderness. Some sag of posterior canal wall and prominence of posterior superior quadrant.
3-3 9:15 A. M.	36.8	82			13,050	2			12	45	30	10	More bulging of canal wall. Discharge continues. Mt. opening smaller. Another paracentesis done on same ear. Mastoid tenderness increased. Pus washed from the right maxillary sinus.
3-4 9:00 A. M.	36.8	76	1,140,000	80	17,500	2	1	14	65	14	6	6	Simple mastoidectomy done. Cortex very soft and acellular. Lateral sinus wall well defined and normal. Cells in retroauricular trough contained pus. Pus and granulations in antrum. Area cleaned, closed with drainage.
3-5 8:00 A. M.	37.6	90			16,200				12	71	11	4	Patient's condition satisfactory. Some occipital headache.
3-7 8:30 A. M.	37.6	80			12,850				6	67	18	9	Canal draining thick mucopus. Postauricular drainage abundant. Considerable pain in right ear. Hemogram approaching normal limits.
3-10 8:30 A. M.	37	80			9,450				5	63	21	10	Continued profuse drainage, examination of which showed moderate number of short chain streptococci.
3-12 8:00 A. M.	37	74			8,200	1			4	62	26	7	Less canal drainage. Less postauricular discharge. Antrum irrigation showed more mucoid secretion.
3-18 9:00 A. M.	36.8	88			8,000	2			5	63	25	5	Ear condition not greatly improved. Pt. feeling much better.
3-22 8:30 A. M.	36.8	86			8,200	3			4	60	30	3	Ear almost dry. Still some postauricular drainage, but less in amount. Hemogram has remained within normal limits for past two weeks.
3-27 9:15 A. M.	36.8	88			8,300	3			5	62	27	3	Ear continues dry. Pt. much improved. Slight postauricular drainage. Discharged; to be further observed in O. P. D. Clinic.

Case 2.—M. D., female, 27 years old, a senior medical student, was admitted to Barnes Hospital March 15, 1930, on account of an acute suppurative otitis media of two days' duration. The patient's past history was important only in that she was seriously ill for a month during October, 1927, with a bilateral colon bacillus pyelonephritis and that she was admitted to the hospital in January, 1930, because of an acute attack of appendicitis. Recovery from the latter occurred, however, without operative interference.

Two days before admission to the hospital the patient was awakened from sleep by a dull, throbbing ache in the left ear. This pain became gradually more severe until morning, when an otologist was called who opened the drum with the release of considerable serum under pressure and the almost immediate relief of the pain.

The patient's subsequent course is summarized along with the hemogram studies.

Comment.—From the hemogram chart it is seen that the stab cells arose alarmingly at the time the right ear became involved, but began to drop immediately after a myringotomy released a marked amount of pressure exudate. In this case the resistance curve remained consistently favorable, despite the high septic temperature, and a theoretical resistance peak occurred about a week before operation, as the blood picture remained practically normal for a few days. Gradually, however, the stab cell count began to elevate again, and at this time clinical observation showed beginning annular periostitis, and bilateral mastoidectomy, now definitely indicated, was promptly carried out, the patient's resistance still being high. The subsequent postoperative course bears out the belief that the optimum time for surgical intervention was chosen.

HEMOGRAM STUDIES—CASE 2.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	My.	Mt.	Stb.	Seg.	Lv.	Mn.	REMARKS
3-15 11:00 A. M.	37	100	4,270,000	80	12,700	1				6	74	13	6	Ft. feeling better; pain in ear less. Urine negative. W. B. C. moderately elevated with increased PMN. %. Stabs low.
3-16 8:00 A. M.	37.6	100			10,420	1				7	52	36	4	Pt. complaining of slight pain in both ears, which by 12 A. M. was quite severe in right ear. Exam. at this time showed a M. T., which was reddened but not bulging. By 8 P. M. the temp. had risen to 400 C. and stab had increased 100%. The pain in right ear was acute. Exam. revealed a definitely bulging, reddened M. T. Myringotomy released a large amount of serosanguinous exudate under pressure.
8:00 P. M.	40	120			11,450	1			1	15	54	19	10	
										70				
3-17 8:30 A. M.	38	112			11,950					11	54	30	5	Temp. still elevated. Pt. quite toxic and exhausted. Some pain in both ears and mastoid areas. Temperature throughout both mastoid areas. Morphine required.
3-18 8:30 A. M.	38	100			12,550	2				7	59	28	4	Total W. B. C. rising; drop in stab forms with appearance of eosinophiles. Culture of left ear reported positive for hemolytic strept. Pt. generally improved and less toxic. Discharge from right ear less profuse and less offensive. No canal sag evident on either ear. Mastoid tenderness extreme on right side, but somewhat less on the left. In P. M. pt. became more restless, complained of chilly sensations with ass'td temp. rise to 39.4 C., and pulse 120. Hemogram unchanged. Urine neg.
6:00 P. M.	39.4	120			13,100	1				8	61	27	3	
										69				

HEMOGRAM STUDIES—CASE 2 (Continued).

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B. Mv.	Mt.	Sub.	Seg.	Ly.	Mn.	REMARKS
3-19 10:30 A. M.	37.8	112			12,350				6	60	27	6	Temp. peaked at 39.4 C. at 4 P. M. Drainage from each ear adequate; no increase in mastoid tenderness and no new symptoms suggestive of further spread of infection. Hemogram shows increasing resistance.
3-20 10:30 A. M.	37.8	112			12,350				6	60	27	6	X-rays of both mastoids show diffuse haziness throughout each pneumatic structure, but no dissolution of intracellular trabeculae. Discharge changing to a frankly purulent character and general amount less. Left mastoid tenderness distinctly decreased. Ophthalmoscopic exam. neg.
8:00 A. M.	37	90			11,100								Pt. menstruating. Has some right ear-ache. Is quite deaf in both ears. Mastoidectomy considered. Pt. resting much better.
3-21 9:30 P. M.	38.2	96			13,200	3	1		5	61	25	5	
3-22 9:30 A. M.	37.2	90	3,820,000	75	12,550	1	1		4	61	30	4	W. B. C. steadily falling and blood picture within normal limits. Mastoid plates still show no bony destruction.
3-25 9:00 A. M.	37	80			11,100	1	1		4	56	31	5	Pt. generally improved. Temp. peak 38 C. Ear pain and mastoid tenderness on both sides much less. Mastoid plates still normal. W. B. C. continues to remain elevated. Much mucus and fresh pus cells with scattered short chain strep. noted in ear sin.
3-27 8:30 A. M.	36.2	80			10,200	2			5	47	38	2	Pt. feeling much better. Practically no mastoid tenderness on either side. Hearing is better and head noises less. Discharge from both ears continues profuse.

HEMOGRAM STUDIES—CASE 2 (Continued).

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	My.	Mt.	Stb.	Seg.	Lv.	Mn.	REMARKS
3-28 8:00 A. M.	37.2	82	4,060,000	80	9,200	5	2			5	53	30	5	W. B. C. continues to drop slowly. Hemogram not elevated. Slight thickening of fundus of each ear canal present. Urine negative.
3-31 8:00 A. M.	36.6	80			9,450	1	1			6	55	33	4	Stab cells gradually increasing and total PMN. % again increasing. Ears continue to drain profusely an discharge is frankly purulent, exam. of which shows less mucus and old disintegrating pus cells with many free, present. Temp. remains about 37.4 C. P. M. back still evident.
4-1 8:00 A. M.	37	100			10,100					7	58	30	5	Discharge from each ear is quite profuse and the fundus of a fullness of all the tissues in fundus of each ear canal. Bilateral mastoidectomy advised. X-ray plates showed definite bony dissolution in both mastoids (esp. left). Hemogram indicates decreasing resistance.
4-2 12:00 A. M.	37.6	108			10,400					7	62	28	3	Bilateral simple mastoidectomy performed. Periosteum over both structures stripped with difficulty. Very large pneumatic cells filled with pus and granulations found on both sides. No lateral sinus involvement. Cultures from mastoid reported pure hemolytic strep.
4-3 8:00 A. M.	37.6	106	3,910,000	80	10,000	1				6	59	30	4	Pt's condition very satisfactory. No post-operative temp. rise. Hearing greatly improved. W. B. C. not elevated and hemogram indicates no further extension of the infection.
4-5 8:30 A. M.	37.2	90			8,200					5	64	29	2	Total W. B. C. rapidly falling. Hemogram approaching normal. Wounds healing well. Both ear canals dry. Convalescence has been uneventful.
4-7 8:30 A. M.	37.4	80			7,800	2				4	57	34	3	Blood picture quite normal. Pt. sitting up and feeling very well. Hearing strikingly improved.
4-11 9:00 A. M.	36.8	76	8,950,000	78	8,000	4				4	50	38	4	Drainage scant and of serous character from both sides. Wounds clean and healing in fast. Hemogram and temp. continue normal. Pt. discharged.
4-30														Pt. has continued quite well.

Case 3.—M. S., white, aged 4 years, had been in good health until February 13, 1930, at which time she complained of malaise, headache and chilly sensations. Three days later, the child's condition was not improved, and there had developed a high fever and a rash over the right side of the neck. A physician was called, and suspected scarlet fever; he had the child transported to the Isolation Hospital, where for the following two weeks she ran a rather typical course of this disease. On the first of March, the patient started to run a temperature of 103.6° F. Examination revealed both ear drums inflamed and bulging, and a bilateral myringotomy was performed. The temperature remained high, and on the 5th of March a typical measles rash with Koplick's spots was found. The child apparently recovered from the measles, but on the 17th of March started to run an afternoon temperature up to 105° F. The following day the urine was found to be loaded with white blood corpuscles and a diagnosis of pyelitis was made. The urine failed to clear under adequate therapy and the temperature remained high. The white count had progressively risen from 5,300 on March 5th to 21,000 on March 17th. X-ray plates of the mastoids showed haziness of the right. The child was brought to Children's Hospital on March 23, 1930.

Physical examination revealed a well developed, slightly undernourished child, somewhat pale and who was well oriented and co-operative. The eyes were normal. Nose—all the mucosa deeply congested and mucopus on the floor of each side. Throat examination showed very large tonsils, subacutely inflamed and the presence of mucopus on the posterior pharyngeal wall. The ears when examined showed both canals filled with thick yellow pus which on the left side came through a posterior inferior perforation and on the right side through an anterior inferior perforation. On each side the openings were inadequate, as both drums were thickened and bulging. There was bilateral mastoid tip tenderness present. The remaining findings were essentially negative and no signs of central nervous system involvement were elicited. Laboratory findings showed red blood corpuscles 4,100,000, white blood corpuscles 5,600, with 65 per cent polymorphonuclear leucocytes and 35 per cent lymphocytes. The urine was

found to be clear with only a trace of albumin and occasional W. B. C. in clumps and without casts or R. B. C.'s. The blood culture was reported positive for hemolytic streptococcus. The temperature was typically septic, varying from 40 to 38° C.

The child was transfused with 150 cc. of blood on the 25th of March; there was no reaction and the patient felt better. X-ray plates of the mastoids showed marked haziness of cell detail on each side. Both drums were widely reincised under ethyl chloride anesthesia and pus under pressure released from each side. On the 29th of March the second blood culture was reported positive for hemolytic streptococci.

At this point daily hemogram studies were begun and the patient's course is included in the summary.

Comment.—In this case two consecutive blood cultures were found positive for hemolytic streptococcus before the bilateral mastoidectomy was performed. It is notable that a differential count using the Ehrlich classification showed almost normal polymorphonuclear and lymphocytic percentages—65 and 35, respectively, with an associated low total W. B. C. count. Hemogram studies shortly afterwards revealed a distinct left shift, further verifying a guarded prognosis. The first postoperative day was quite satisfactory until late in the afternoon, when the temperature suddenly rose to 40° C., with a pulse of 160. The blood picture at the peak of the temperature rise showed only a rise of 2,000 cells but over a 100 per cent increase in the stab cells and an associated polymorphonucleosis, lymphopenia, monocytopenia and eosinophilia—all pointing to a rather sudden and fulminant extension of the infection. The patient was examined at the time for meningeal or pulmonary signs, but the latter were negative and only a questionable stiffness of the neck and a diffuse abdominal tenderness were clinically manifest. The following morning the white blood count had dropped 1,000, but the total stab cells and polymorphonuclear percentage were still mounting. Examination now revealed signs of meningitis, further verified by the demonstration of over 1,500 polymorphonuclear cells and many streptococci in the spinal fluid.

While cisternal drainage caused a definite clinical improvement for a short time, the blood picture continued to show a

regenerative shift despite a slowly falling total white count. The morning of the patient's last day, the stabs became markedly increased and myelocytes appeared in the peripheral blood stream—such could only be interpreted as denoting a fatal outcome. This case is illustrative of the value of the hemogram in determining the onset of untoward complications. The blood picture definitely showed that such had occurred before clinical confirmatory signs appeared.

HEMOGRAM STUDIES—CASE 3.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	Mx.	Mt.	Std.	Seg.	Ly.	Mn.	REMARKS
3-26-30 8:30 A. M.	39.4	120	4,054,000	80	10,300	1	1	1	1	18	42	25	10	W. B. C. increased. Marked shift to left, indicating regenerative response. Cont'd. ear discharge
3-27 8:30 A. M.	37.4	125			15,600	2	2			11	26	42	17	W. B. C. rising though total stabs are lowered. Pt. continues to show septic reaction.
3-28 8:30 A. M.	38.4	130			15,100	1	1			11	43	30	14	Ear discharge pulsating and canal sag bilaterally present. Eye grounds normal.
3-29 8:30 A. M.	37.6	125			16,180	4	1			12	48	26	9	W. B. C. still rising. Bilateral mastoidectomy performed. Pus and granulations found on each side, left lateral sinus discolored and opened at site of entrance of mastoid emissary. Free bleeding obtained from both ends. Packed above and below. Child transfused.
3-30 8:00 A. M. 3:30 P. M.	40	140	4,060,000	80	18,200				1	26	57	11	3	Child's condition quite satisfactory in A. M. Temp. steadily declined to 36.5° C. W. B. C. 2,000 higher than before operation. Stabs and cells increased 100% with a much higher PMN. total. Chest neg. No abnormal CNS findings except neck rigidity.
3-31 8:00 A. M.	41	160			17,200				1	27	62	9		Cont'd marked regenerative shift. Eye signs neg. Marked rigidity of neck, bilateral Kernig and Babinski. KK's and AJ's hyperactive. Abdomen tender and distended. Spinal fluid quite cloudy; cell count 1,500 with 48% PMN. Smears showed many Gram-short chains of streptococci. Cerebral drainage done in P. M.—large quantity of fluid liberated when dura opened. Returned to ward in good condition.

HEMOGRAM STUDIES—CASE 3 (Continued).

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	My	Mt.	Stb.	Seg.	Lv.	Mn.	REMARKS
4-1 8:15 A. M.	38.3	130	3,890,000	70	13,900			1	1	18	58	18	4	W. B. C. slightly lower, with ass't'd fall in stab count. 120cc. whole blood transfusion given. Temp. raised from 39-40.5 C. Drainage profuse. Child slightly improved.
4-2 4:00 P. M.	40.4	140			15,650			1	2	25	35	28	9	W. B. C. increasing and stab cells definitely elevated. Temp. still 39-40 C. despite sponges. Child more irritable.
4-4 11:00 A. M.	39.4	140	3,850,000	65	14,800			1	2	18	53	19	7	Pt. somewhat better, takes fluids in increasing amounts. W. B. C. lower; sudden rise of lymphocytes.
4-5 8:00 A. M.	39.4	140			14,350				4	25	50	25		Shift to left again more pronounced. Temp. peak 40 C. Kernig still positive.
4-6 8:00 A. M.	38.4	150			12,550				3	22	56	18	1	Drainage much less and continued scant after reestablishment of flow. At 4 P. M. pt. had sudden onset of generalized convulsions with ass't'd frothing and drooling from the mouth. Temp. 40.5 C. Convulsions controlled with sodium luminal and chloroform.
4-7 9:00 A. M.	40	170	4,000,000	60	11,750			2	6	38	33	15	6	W. B. C. falling in face of an increasing hyperplastic reaction. Pt. unconscious thru-out the day and died at 7:55 P. M.
														Autopsy was not obtained.

Case 4.—L. H., white, female, aged 39 years, was admitted to the Jewish Hospital March 2, 1930, because of a discharging left ear for the past ten days. The patient had been in good health up to the morning of March 20, 1930, at which time she noted a sudden severe pain localized in the left ear. No previous sore throat or upper respiratory infection had been noted. Oil drops were placed in the ear and a hot pad applied. During the night the pain suddenly ceased and the patient noted that the left ear was discharging a thin, bloody exudate.

The ear continued to discharge freely, and by the second day after the spontaneous rupture the exudate had become purulent in character and pain was again experienced. An otologist was called at this time and found the perforation of the drum inadequate for free drainage. Under ethyl chloride anesthesia a wide myringotomy was performed, after which the discharge continued to be quite profuse and purulent. For the following seven days the patient was without pain and never had more than one degree of temperature elevation daily. The following day, however, the pain became more marked and the temperature rose to 102° F. Re-examination of the ear showed a very profuse, pulsating discharge coming from a posterior inferior perforation. Both tonsils were noted to be subacutely inflamed and the temperature rise was attributed to this. X-ray plates of both mastoids, taken on April 1, 1930, showed a marked haziness on the affected side—a diploetic type of mastoid structure being present. A white blood count that day was 9,800. The patient was brought to the hospital the following day (April 2, 1930) for further observation. At this time the sore throat was severe.

Admittance temperature was 101° F., pulse 104, respirations 22. The left external auditory canal was filled with a pulsating, yellowish discharge which when cleared away revealed a tympanic membrane perforated in the posterior inferior quadrant, loss of drum markings and a slight fullness in the fundus of the canal. There was a slight left mastoid tip tenderness. The pharynx was reddened and the tonsils reddened and submerged.

Urinalysis was negative. The patient's subsequent course is summarized along with the hemogram studies.

Comment.—The distinct benefit derived from serial hemograms is well shown in this case. Variations in the blood picture were at no time very marked, and, once the individual resistance reaction was established, even the often slight variations were significant. It is notable that the stab cell count remained slightly elevated, even when the remaining clinical picture pointed to a healing process and though the patient was given every conservative consideration it was found necessary to establish postauricular drainage. This done, the blood picture rapidly resumed its normal state. As in Case 2, operative interference was performed at or near the patient's resistance peak.

HEMOGRAM STUDIES—CASE 4.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B. MY.	Mt.	Stb.	Seg.	Ly.	Mn.	REMARKS
4-2 9:00 A. M.	101	100	4,420,000	85	10,250	2			12	69	15	2	Moderately elevated W. B. C. count; increased PMN. %. Distinct regenerative shift.
4-3 9:15 A. M.	99	88			11,200	3			10	53	24	10	Definite fall in PMN. % with lower stab count despite rise in total W. B. C. Pt. feels better. Fundus fullness in left ear. An early coalescent form of mastoiditis.
4-4 8:45 A. M.	98	90			11,700	1	2		10	52	26	8	Discharge more profuse; beginning sag of superior canal fundus. Discharge shows many Granuloploccoid and short chain strep. Pus cells actively phagocyt.
4-5	100	88			11,100	2	1		8	55	29	5	Definite fall in total W. B. C. and PMN. % increasing eosinophils and lymphocytes.
4-6	99	90			9,400	4			8	45	33	10	Discharge from ear still profuse, purulent and pulsating. Superior canal sag still present but not increased. X-rays show no definite cellular destruction.
4-7	98.6	84			9,500	2	1		9	48	36	4	
									57				
4-8	98.6	82			9,700	3			9	59	22	7	In the past three days temp. has remained practically normal; W. B. C. falling.
4-9	98	92			9,400	2			8	61	25	4	Hemogram shows increasing resistance to infection, but has not returned to normal limits. Pt. feeling much better. Ear discharge, almost ceased. Loss of fullness of canal fundus. Pt. discharged; to be followed at home.
4-10	98	80			8,840	4			7	52	33	4	
4-11	98.6	82			8,200	3			7	50	33	4	
									57				

HEMOGRAM STUDIES—CASE 4 (Continued).

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	My.	Mt.	Stb.	Seg.	L.V.	Mo.	REMARKS
4-16 9:50 A. M.	99	88			10,700					9	63	23	5	Pt. re-enters hospital as discharge is again profuse, and superior canal sag is much more manifest. Total W. B. C. have increased nearly 2,000, PMN. % again elevated though stab cells remain as before.
4-17	99	80			10,600	1				10	54	31	4	Patient's hearing in left ear definitely impaired. X-ray plates show a slight but definite increase of bony destruction around the antrum. Hemogram continues to show an increasing infectious response.
4-18	98	84			9,800					8	50	32	8	
										58				
4-19 8:40 A. M.	98.6	90			10,800					8	56	30	6	Left simple mastoidectomy performed; Perosteum stripped easily; thick cortex removed disclosed displace cell structure filled with pus and granulations, most marked around the antrum. Lateral sinus normal. Closure with drainage.
4-20	98.8	96		4,400,000	85					6	63	29	3	Postoperative course exceedingly uneventful. Temp. at no time rose above 99 F. W. B. C. increased but was not followed by stab elevation and blood picture by the second P. O. day had fallen to normal. Ear wound healing fast.
4-21	98	84			9,800	1				5	60	30	4	Healing quite improved. Discharged.
4-26	98	80			7,350	3				4	57	32	4	
										61				

Case 5.—H. R. S., white, aged 32 years, entered Barnes Hospital on March 1, 1930, with a diagnosis of acute purulent otitis media (right) of five days' duration.

Since early childhood the patient had had numerous attacks of otitis media (bilateral). The drums would rupture and the aural discharge continue for intervals varying from two weeks to three months. The right ear gradually was noted to be defective as regards audition.

Ten days before entrance into the hospital the patient developed a sore throat with associated general weakness and frontal headache. There had been a slight fever at first, but this gradually mounted to about 38° C. on the fifth day of the disease, when a physician was called because of severe pain in the right ear. The temperature continued between 38 and 40 degrees C., and X-ray plates, taken on April 30, 1930, showed a bilateral chronic mastoiditis. The patient became more toxic and was advised to enter the hospital.

Physical examination revealed a well developed, well nourished male whose sensorium seemed slightly dulled and who was rather noncommittal to questioning. The temperature was 38.6° and pulse 108. The eyes were within normal limits (including fundus examination). The nose showed some redness and congestion of the mucosa.

The external canal of the right ear contained a creamy exudate with no perceptible odor. The right drum was reddened, thickened and showed a posterior inferior perforation. The left ear canal was dry. There was no mastoid tenderness over either side. The lungs upon percussion were resonant throughout, except the right base posteriorly. Occasional moist râles were heard here and there throughout the chest, and in the right posterior base persistent fine râles, increased by cough, were noted.

A culture of the discharge from the right ear was reported positive for hemolytic streptococcus and staphylococcus aureus.

The patient's subsequent course is tabulated along with the blood findings.

HEMOGRAM STUDIES—CASE 5.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B. Mv.	Ml.	Stb.	Seg.	Lv.	Mn.	REMARKS
4-1 5:30 P. M.	39.4	108	4,790,000	96	13,850		2	24	60	10	4		Pt. feeling fairly well. Is somewhat sluggish mentally. Hemogram shows a decided regenerative shift.
4-2 8:00 A. M.	38.4	108			15,800		1	19	62	11	5		W. B. C. increased, left shift less prominent though still elevated. Pt's general condition improved. Right ear draining a thick yellow exudate. Left ear quiescent.
4-3 8:00 A. M.	38	100			14,800						16	2	Pt. quite toxic and sluggish. Vomits all fluids except water. Chest clear except for a few sonorous rales which disappear upon coughing.
4-4 7:45 A. M.	38.7	102			18,500		2	16	65	13	4		W. B. C. increased 4,000, hemogram continues to indicate that infection is unduly severe. Discharge from right ear is scant.
4-5 A. M.	38.2	96			17,400								Stained smear unsatisfactory for differential. Pt. continues somewhat toxic.
4-7 8:15 A. M.	39	108			21,100			16	61	19	4		W. B. C. rising also P. and T. Ear condition improved. At 3 P. M. had a sudden attack of persistent vomiting, projectile in type and unrelated to previous food ingestion. Temp. rose to 40.4 C.; as'd severe chill and slight headache. No definite signs of pulmonary involvement made out. Both eye grounds showed blurred and hyperemic discs but not suggestive of increased intracranial pressure. No signs of meningeal involvement elicited. Ear unchanged.

HEMOGRAM STUDIES—CASE 5 (Continued).

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	MV.	ML.	Stb.	Seg.	Ly.	Mn.	REMARKS
4-8 8:30 P. M.	38.8	110	4,880,000	85	20,800	1			16	64	16	3		During the night pt. developed a severe pain in the right chest. Exam. showed rapid resp. with an expiratory grunt; marked limitation of excursions on right side. X-ray shows an area of density at base of right lung, wedge shaped and standing clear of diaphragm. Diagnosis of pulmonary infarct made.
4-9 6:00 P. M.	38.6	130			21,700			626	18	63	13	4		Cont'd severe infection with W. B. C. total rising. Strapping of right chest relieved pt's pain considerably. Blood culture reported negative. Impression of pulmonary embolism from a lateral sinus thrombus.
4-11 8:30 A. M.	38.8	124			26,850			1	16	70	9	3		W. B. C. and total PMN. % still rising. Pt. is toxic, respirations rapid with expiratory grunt. Face is pale, anxious expression. No physical signs in chest. X-ray shows an almost complete opacity of right lung field. Diagnosis of pulmonary infarction and hydropneumothorax. Sputum blood streaked. Mouse injection for pneumococci negative.
4-12 8:00 A. M.	39	124	4,600,000	85	34,200	1		2	23	66	6	2		W. B. C. increased 8,000, hemogram shows marked regenerative-degenerative shift. Moderate cyanosis of lips and fingers. Percussion note over lower half of back and right axilla dull. Dullness present over remainder of right chest. Ear condition practically subsided.

HEMOGRAM STUDIES—CASE 5 (Continued).

Date	T.	P.	R. B. C.	Hb.	W. B. C. E.	B. My. M.	Stb. Seg.	L.V. M.	REMARKS
4-14 8:00 A. M.	38.2	130			30,000	1	2 18 71	71	1 Pt. somewhat improved. Dullness persists in right lung base with bronchial breath sounds and whisper.
4-15 8:00 A. M.	39	114			33,800		1 17 75	6	1 W. B. C. count continues elevated. Very high total FMN. %. Pt. irritable at times. Flatness and diminished tactile fremitus suggest fluid in chest. Right ear again draining thick yellow pus; drum not bulging.
4-16 10:00 A. M.	39	140	44,476,000	85	42,600 48,150		1 18 70	5	3 W. B. C. total increased 9,000 with increasing regenerative-degenerative shift. Pt. in considerable distress; moderately cyanotic. No changes in physical signs of chest. Is 9th day since onset of pain in chest and consolidation.
5:00 P. M.							6 22 66	3	1 Pt. much worse, cyanosis deeper, resp. very labored and rapid. Pulse rapid but of good quality. B. P. 100/60. O ₂ insufflation began. Chest signs show increased area of consolidation with many rales and rhonchi. W. B. C. markedly increased, all leucocytes immature.
4-17 8:00 P. M.		142			56,150		7 25 64	3	1 Cyanosis somewhat controlled with tent. Pulse steadily rising while temp. falling. Resp. labored. Urine shows tr. of alb. + + + + W. B. C. + R. B. C. galac faintly +.
4-19 8:00 A. M.	37.5	146			56,900		8 24 63	4	1 Pt. definitely worse. Heliotrope cyanosis has returned. Pt. delirious and talks considerably despite increased dyspnea. B. P. 140/70. Abdominal distension increased. Blood culture positive for streptococcus. W. B. C. almost 60,000. Hemogram now definitely degenerative. Patient died 11:17 P. M.
4-20 9:00 A. M.	38.2	146			59,600		2 6 24 61	6	1 93

Postmortem findings were quite significant. The right mastoid revealed very small diploetic cells which were not definitely involved macroscopically. The lateral sinus on this side showed an occlusive thrombosis limited to the knee; jugular bulb was not involved. The lungs presented a massive empyema present in both lower lobes. The parenchyma was diffusely "riddled" with many infarcts. There was some pericardial effusion and the kidneys showed signs of hemorrhagic nephritis. It was believed that the lateral sinus thrombus had been present for some time as a mural clot and became occlusive late in the course of the illness. The cause of death was attributed to the multiple septic infarctions of the lungs, caused undoubtedly from the focus in the sigmoid sinus.

This case is illustrative of the complications that often occur due to an acute exacerbation superimposed on an old chronic mastoiditis. While the total W. B. C. count was only moderately elevated (13,850), the Schilling hemogram showed a severe shift to the left, much more than could be accounted for with an acute mastoiditis simplex. The total count at first began to increase slowly, the stab cells remaining elevated but not progressively so until frank signs of pulmonary involvement were observed. As the total cells rapidly increased, a further left shift became evident by the increasing juvenile forms, and at the last, myelocytes appeared in the peripheral blood stream. The patient exhibited a magnificent resistance to the infection, as can be seen by the marked total W. B. C. count and the consistently high polymorphonuclear percentage (89-96), of which the segmented forms were much higher than would be generally expected, especially when viewed in the light of the process revealed at postmortem. It was believed by some that the lateral sinus thrombus was present even before the patient entered the hospital. Such an explanation would coincide with the hemogram interpretations which, as was pointed out, gave reason to believe that from the first a complication of some sort was present.

Case 6.—C. L. P., age 3 years, was admitted to the Children's Hospital on April 8, 1930, with a diagnosis of right otitis media, right mastoiditis and acute tonsillitis. Family and past history were unimportant. The child's present illness began two weeks previously, at which time there was noted a sore throat, slight fever, and associated swollen cervical nodes. On March 30, 1930, the patient developed a high fever and an otolaryngologist was called in consultation, who performed a myringotomy of the right ear. The ear discharged profusely and the temperature continued elevated—99 to 100° F. for the following week, and then rose to 102° F. with an associated chill lasting 20 minutes. The right ear continued to be painful, so the child was brought to the hospital.

Physical examination showed a well developed but slightly undernourished boy, quite pale but in no acute pain or distress. The eyes were negative. The right ear canal was freely discharging a profuse purulent exudate which, when removed, revealed a posterior inferior perforation but no upper posterior quadrant sag. The left ear was normal. The nose showed a slight bilateral mucopurulent discharge. The tonsils were large, red and cryptic with a caseous exudate in the crypts. The posterior and anterior cervical lymph nodes were bilaterally enlarged but not tender. The chest, abdomen and extremities were essentially negative.

The patient's course is included in the hemogram summary.

Comment.—While the patient's clinical manifestations at first were quite suggestive of a severe otitic infection—possibly a lateral sinus thrombosis—serial hemogram studies early showed that while an infectious condition certainly existed in the ear, all indications were that there was present an adequate resistance of the patient and the infection was subsiding. The subsequent uneventful course confirmed this interpretation of the hemogram.

HEMOGRAM STUDIES—CASE 6.

Date	T.	P.	R. B. C.	Hb.	W. B. C. E.	B.	My.	Mt.	Sub.	Seg.	Lv.	Mn.	REMARKS
4-8 4:00 P. M.	40.6	130	4,800,000	90	12,200			1	17	52	29	1	Child restless and complaining of pain in right ear. Temp. sponge brought fever down from 39.8 C. to 38 C. Urine negative. Hemogram shows distinct shift to the left.
4-9 4:00 P. M.	37.8	96	4,630,000	90	12,900	1	1		10	60	22	6	W. B. C. continue at the same level as do the total RBC. % but the shift to the left is definitely lowered and eosinophilia is present—all indicating adequate resistance to a subsiding infection. Temp. approaching normal.
4-10 4:30 P. M.	37.6	90			12,300	5			8	41	42	4	Hemogram shows a very definite resistive shift to the right. Temp. continues to remain near normal. Tonsillitis has almost disappeared. Mastoid X-ray plates show no evidences of cellular destruction. Pt. feels well.
4-12 8:30 A. M.	37.4	92			10,800	3			6	40	45	5	Ear discharge changing to a thick scanty mucoid character. Temp. has continued to stay below 38 C. Hemogram nearing normal limits. Pt. discharged.
4-30													Child has continued well. Ear no longer discharging.

Case 7.—M. A. R., white, aged 6 years, was admitted to the St. Louis Children's Hospital, April 3, 1930, with a diagnosis of bilateral otitis media with high fever. The family and past history were irrelevant to her present illness, with the exception that for the past year the patient had several attacks of tonsillitis. The child was in good health until two weeks before entry in the hospital. At this time she first became fretful, fatigued, lost her appetite and apparently was suffering from a slight upper respiratory infection. Two days later she developed a high fever and complained of pain in both ears—the latter rupturing spontaneously and draining a profuse mucopurulent exudate. An otolaryngologist was called and performed a bilateral myringotomy to insure adequate drainage. The child improved for a week and then on April 1, 1930, her temperature suddenly elevated, definite chills being associated with the afternoon temperature rises. On April 3, 1930, a pediatrician, called in consultation, found the patient's urine to be typical of an early hemorrhagic nephritis. A blood count at this time was reported to be W. B. C. 35,000, with 90 per cent polymorphonuclears, 1 per cent basophiles, 4 per cent lymphocytes, 5 per cent transitionals. The ears continued to show a profuse discharge, and the child was brought to the hospital for careful observation.

Physical examination revealed a well nourished and developed child in no apparent pain. There was a bilateral seromucoid discharge from both ears. There was no mastoid tenderness. The tonsils were large and inflamed. Heart, lungs, abdomen and extremities were essentially negative. Laboratory examination showed a urine containing a trace of albumin and occasional clumped white blood cells and a few red blood cells and casts.

Daily hemograms were made on the patient and the course of her illness is included in the chart.

Comment.—As in Case 5, there was a suggestion of serious otitic complications. Blood studies revealed a consistently elevated white count but at no time did the stab cells indicate any further progression of the otitis. The clinical aural symptoms and signs gradually subsided and the patient had an uneventful recovery.

HEMOGRAM STUDIES—CASE 7.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B. Mv.	Stb.	Seg.	Lv.	Mn.	REMARKS
4-3 4:00 P. M.	39.2	108	4,500,000	80	16,500					21		Regular differential. Blood culture neg. Pt. feeling much better.
4-4 4:00 P. M.	39.2	100			15,450					18	2	Regular differential. Pt.'s general condition good. Lungs clear. Urine shows a faint trace of alb.; few R. B. C. and casts.
4-5 8:15 A. M.	37.2	80			14,880	2	1	8	66	17	6	Total W. B. C. falling as in total PMN. No present evidence of severe infection. Ear discharge is profuse. No mastoid tenderness.
4-6 8:30 A. M.	37.4	78			15,300	2		8	59	28	3	Cont's rise in lymphocytes and fall in PMN. An increasing resistance to a subsiding infection. Ear infection markedly improved. Urine only shows a few scattered W. B. C., R. B. C., and casts with alb. neg.
4-7 8:30 A. M.	37.3	74			15,150	2		7	63	24	4	Condition unchanged.
4-8 8:55 A. M.	37.2	74			15,100	3		6	48	35	7	Slight elevation of temp. in afternoon. Continued elevation of W. B. C., but hemogram approaching normal limits.
4-9 9:00 A. M.	37.2	82			14,350	2	1	7	49	37	4	Pt. feels very well. Urine essentially neg.
4-10 5:30 P. M.	36.8	100			14,100	2		6	47	39	6	Continued increasing resistance to infection. Ear discharge changed to heavy mucoid type scanty in amount. Patient discharged.

Case 8.—I. D. N., white, aged 24 years, a senior medical student, was admitted to Barnes Hospital, March 31, 1930, because of pain and tenderness behind the right ear. The family and past history were irrelevant. The patient's present illness began about 1:00 p. m., March 30, 1930, at which time he noted a gradually increasing pain and tenderness behind the right ear, extending a short distance down the neck. The postauricular glands on the same side were enlarged and tender to touch. By evening the postaural pain had become localized more over the mastoid region; there were several chills and the patient felt "feverish." The following morning the pain was more severe and persisted despite rather large and frequent doses of aspirin. There was a distinct redness and swelling present behind the ear, and the otolaryngologist who was called in advised hospitalization for closer observation of the condition.

Positive physical findings were limited to the head. There was a small amount of mucopus in the right side of the nose with associated congestion. The throat was quite normal. The left ear canal and tympanic membrane appeared normal. The right ear canal was red and injected in its posterior portion. Schrapnel's membrane was distinctly reddened and swollen, with prominence of the blood vessels. The drum was not bulging, and while somewhat pink throughout still retained its normal luster. There was definite tenderness and redness over the tip of the right mastoid. The chest, abdomen and extremities were essentially negative.

The patient's subsequent course is tabulated with the hemogram studies.

Comment.—While the patient's symptoms were at first quite suggestive of mastoid involvement, the fact that because the W. B. C. count and temperature suddenly elevated indicated further extension of the process was not borne out by hemogram studies. The stab cells remained relatively low and unchanged for a short time, then rapidly dropped to near normal. In this case a lymphadenitis and periostitis of unknown origin apparently were the cause of the clinical manifestations. The condition subsided almost as quickly as it appeared.

HEMOGRAM STUDIES—CASE 8.

Date	T.	P.	R. B. C.	Hb.	W. E. C.	E.	B. My.	Mt.	Sub.	Seg.	Ly.	Mn.	REMARKS
3-21 8:30 P. M.	37.3	100	5,110,000	90	11,400		1		10	62	25	2	Pt. feeling somewhat better, yet still has considerable local pain. Hemogram shows only a moderate infectious response. Urine neg.
4-1 8:00 P. M.	38	104			10,300		1		8	60	28	3	Right M. T. more reddened and slightly bulging. Post-articular tenderness more diffuse with associated redness. X-ray plates show diffuse changes throughout both pneumatic mastoid regions but no cellular dissolution.
4-2 8:00 A. M.	38.2	100			13,150	1		1	10	76	8	4	Local condition much improved. Infection of the vessels on right M. T. has disappeared. Left M. T. slightly injected. Swelling and tenderness over right mastoid and cervical adenitis less pronounced. Pt. more comfortable in A. M. but less so in P. M. Temp. 39 C.). Hemogram shows no further increase of infectious process.
4-4 8:00 A. M.	36.4	90			16,700	4	2		10	44	36	4	W. B. C. falling fast with ass't'd definite healing response. Pt. feels much better. Temp. subnormal all day.
4-5 8:00 A. M.	36.6	86			9,800	2			6	50	40	4	The cervical adenitis which apparently was responsible for the local condition has entirely disappeared, though not eradicated. Hemogram nearing normal limits. Pt. discharged.
													Pt. has continued well.

Case 9.—C. F., aged 5 years, was admitted to the Jewish Hospital March 31, 1930, with a diagnosis of right acute otitis media and mastoiditis. The patient had been for the last year under the care of the Central Institute for the Deaf, where his condition of congenital aphasia was being studied. Past history is important in that the child has always been incontinent of urine and feces and that even a mild upper respiratory infection always provoked a marked febrile reaction (upwards to 105°).

The patient's present illness began two weeks before admission into the hospital, at which time he developed an acute otitis media in the right ear, necessitating myringotomy. Two days before admission he had a temperature elevation ranging from 102 to 104° F., with swelling and tenderness over the right mastoid area. The fever continued quite high and he was sent into the hospital for operation.

Physical examination was essentially negative with exception of the local ear findings. The right ear showed a posterior canal wall sag and a bulging drum. The left ear was within normal limits. Urinalysis was negative and W. B. C.'s were 19,600, no differential being made. A right simple mastoidectomy was performed the same day of admission. No pus or granulations were found in the mastoid cells. The dura and sinus were not exposed and the wound was packed open with iodoform gauze. A slight, right facial paresis was noted when the bandage was applied.

For the following six days the temperature continued to remain elevated and the child seemed to have diminished resistive and recuperative powers. At the first dressing the wound was clean and the ear was dry, but on the sixth postoperative day it was noted that both ear canals were draining purulent material, culture of which was reported as containing hemolytic staphylococcus aureus. General examination revealed nothing to account for the toxicity, and there were no signs of lateral sinus thrombosis. The temperature continued elevated and of septic character. Daily hemogram studies were started the following day and the patient's subsequent course is summarized in the chart.

Comment.—In this case it is noted that the stab cell count remained consistently elevated, pointing to continued infection, and which later appeared as a left ear involvement. Even when the latter had subsided, however, and the temperature had remained flat for six days, the stab cells still did not correspondingly fall. After the second flare-up, for which no definite cause could be found, subsided, the child has continued quite well, but the hemogram, while showing less left deviation, is still elevated and the mastoid wound is slowly healing.

HEMOGRAM STUDIES—CASE 9.

Date	T.	P.	R. B. C.	Hb.	W. B. C.	E.	B.	My.	Mt.	Stb.	Seg.	Lv.	Mn.	REMARKS
4-8 7:30 A. M.	101.4	126	3,690,000	70	15,100				2	18	52	24	4	Temp. peak 104.4 F. at 8:30 P. M. General condition improved. Less pus in ears.
										72				
4-9 7:45 A. M.	102.2	130			14,250				1	16	48	26	9	Slightly lower total W. B. C., PMN, % and stab cells. Wound clean. Cont'd left deviation.
										65				
4-10 7:45 A. M.	100	106			16,200	4				16	47	27	4	Temp. still elevated though peaking lower. Right facial paresis continues.
										63				
4-11 7:40 4-12					15,300									100 cc. whole blood transfusion.
	100	110	4,770,000	85	14,300	1			14	50	30	5		Temp. elevation to 104 F. after transfusion returned quickly to normal.
										64				
4-13 7:40	99.2	128	4,380,000	85	12,100	1	1		11	54	29	4		Stab forms dropping as is total W. B. C. Patient's condition unchanged.
										65				
4-14 7:50 A. M.	101	92			10,000		1		11	46	34	5		Poor ratio between stabs eosinophiles and lymphocytes. Wound healing slowly. Continued discharge from ear.
										57				
4-15	100.4	120			10,400	2			9	46	38	5		Temp. remains only slightly elevated. Pt. feeling much better.
										55				
4-18	99.4	110	4,310,000	80	11,400	1			1	14	44	32	3	Unexplained shift to the left as evidenced by increase in stab cells.
										57				
4-19 7:55					10,800	1			12	47	36	4		Temp. rose to 102 F. Profuse discharge from left ear with some asst'd mastoid tenderness.
										59				

HEMOGRAM STUDIES—CASE 9 (Continued).

4-20 8:00 A. M.	99	106	3,860,000	75	10,400	2	16	43	34	6	R. B. C. falling. Left shift more prominent. Temp. rose to 105 F. Left ear rein- closed.
4-22	98.6	100			9,800	2	15	48	32	3	Temp. near normal. Still elevated stab cells. Urine neg. No malarial parasites.
4-23	98.6	100			9,700	3	11	46	36	4	Count and total stabs slowly dropping. Blood culture neg.
4-25 7:30 A. M.	99	98			9,600	3	10	47	38	2	Child apparently getting well. Temp. flat for 6 days. Hemogram still shows left.
4-26 8:00 A. M.	99.5	100			10,300	1	11	49	37	2	Sudden elevation of temp. to 105 F. de- scribe very careful search; no local or ge- neral condition found to explain rise. Child quite prostrated; questionable meningeal signs. Chest and urine neg.
4-28	99.2	104	3,970,000	75	11,500	3	12	41	35	3	W. B. C. slightly increased; temp. again normal. Still no evidence of what caused flare.
4-30	98.6	106			9,200	3	1	9	44	3	W. B. C. falling slowly. Increased eosin- ophiles and lymphocytes sl. rt. shift.
5-3 8:30 A. M.	98.6	98			8,750	2		9	47	35	7
5-5 8:00 A. M.	99.2	104			9,400	4	1	11	34	47	3
5-9	98.6	100	3,910,000	80	9,800	3		45	10	54	2
								64			Temp. has remained flat. Wound still draining. Slight odor. Neurological con- sult found no evidences of intracranial in- fection. Pt. looks quite well.

DISCUSSION.

The literature on the subject in question is rather sparse. In a quite recent article Connor⁷ has reviewed the outstanding articles by various observers and includes only eight such reports. The Schilling differential has been used as a routine procedure in comparatively few institutions, and in this country its consistent use as a laboratory aid in otologic conditions has been limited. The work of Kopetsky⁸ and Weiss,² at the Beth Israel Hospital, is the most complete and informative, particularly as regards the value of complete blood studies in the diagnosis and prognosis of the surgical diseases of otology.

The repeated estimation of the red blood cells and hemoglobin gives definite information, especially in cases of severe sepsis. Kopetsky states: "In a patient who presents an acute purulent otitis media with associated septic temperature and severe prostration from the onset, a daily decrease of the number of red blood cells and percentage of hemoglobin speaks for a diagnosis of a hemorrhagic type (hemolytic streptococcus, usually) of mastoiditis and indicates, where all other conditions have been eliminated, an immediate mastoidectomy." Careful check of the red blood cell count and hemoglobin percentage is also of value in suspected cases of lateral sinus thrombosis, as a continued decrease of each, especially after mastoid operation, is very suggestive of further extension of the process into the lateral sinus. The above author also makes the dictum that in cases of acute coalescent mastoiditis a daily study of the red blood cells and hemoglobin subsequent to operation will always lead to an earlier detection of a sinus thrombosis where a hemolytic organism is the offending factor. In the series of nine case studies, the red cell and hemoglobin determinations were not followed so closely as the white cell and differential findings. Six of the nine cases showed positive hemolytic streptococcus ear cultures. Cases 3 and 5 were examples of extreme sepsis. In Case 3 a hemolytic streptococcus septicemia existed shortly after the child became ill with scarlet fever, and repeated blood cultures throughout the illness were reported positive. The red cells and hemoglobin in this case progressively decreased, despite frequent transfu-

sions. Case 5, however, never showed marked reduction of the red blood cells or hemoglobin, and postmortem findings revealed a septic lateral sinus thrombosis which apparently existed for some time. None of the other cases showed definite changes.

The total white count and percentage of neutrophiles vary so greatly among individuals, even with the same type of infection, that it is indeed difficult to establish average figures. Wicart⁹ gives the following values in aural sepsis:

	W. B. C.	% PMN.
Acute suppurative otitis media.....	6-10,000	70
Acute suppurative mastoiditis.....	10-12,000	72
Thrombophlebitis	20-25,000	92-95
Circumscribed meningitis	20-25,000	94
Diffuse meningitis	20-25,000	92-95
Cerebral abscess with encapsulation....	20,000	88-95

Coates, Ernser and Peresly,¹⁰ in an extensive review of 644 cases, tabulate the various white blood cell counts preoperatively and group patients in various classes as determined by the elevation of the count. In about 50 per cent of the 274 cases the W. B. C. count ranged anywhere between 10 and 16,000. Bilateral mastoid involvement showed a higher count. It was felt by these observers that where there was a blood count of over 18,000, there was either a lack of localization, or, on the other hand, a marked body reaction to infection. It was also found that the operative indications usually bore out the policy that it was preferable to wait until there was some evidence of localization, especially as evidenced by the lowering of the total leucocyte count and an increase of the polymorphonuclear cells.

In 259 cases that were studied from the point of increase in polymorphonuclears, 31 per cent showed a polynucleosis between 61 and 70 per cent, 34 per cent between 71 and 80 per cent, and 16 per cent between 81 and 90 per cent. It was noted that the polymorphonuclear count "seems to be of especial interest as an indicator as to when to operate."

In the thirty other uncomplicated acute mastoid cases studied the average white blood cell count ranged between 10,000 and 15,000 and the polymorphonuclear percentage from 65 to 80.

In cases of sepsis and other complications the counts varied greatly. As an illustration, Case 3, in which the child had hemolytic streptococcus mastoiditis, septicemia and meningitis, the highest total W. B. C. count was 18,500, with 90 per cent polymorphonuclears, which progressively fell and at the last, when the infection was at its height, the W. B. C. were 8,300 and the polymorphonuclears 67 per cent. In Case 5, the total W. B. C. count progressively increased from 13,850 to 59,600 at the time of his death and the total polymorphonuclear percentage continually elevated from 83 to 96 per cent. In Case 2, even with a fulminant bilateral hemolytic streptococcus mastoiditis, the highest W. B. C. total was 13,200 and the polymorphonuclear percentage 80, the latter averaging around 69 per cent.

It has long been observed that the total W. B. C. and polymorphonuclear percentages are far from being infallible indicators of the patient's resistance and degree of severity of the infection. More recent studies have emphasized the fact that in many cases the polymorphonuclear percentage and total leucocyte count do not follow each other or the clinical course of the disease. While no laboratory procedure per se can be expected to clearly mirror the reaction of the body forces against various invasive pathogens, yet it is felt that the Schilling differential offers a much more accurate and adequate conception of the latter—particularly in complicated cases, than can be obtained by the older Ehrlich classification of the white blood cells. Kopetsky⁸ states, furthermore: "I have found this method of counting the white cells of inestimable value in determining the procedure to be followed in the so-called borderline cases. Where a mastoiditis is presented and one is in doubt as to whether to operate or not, the staff cells will furnish an excellent guide in helping one to reach a decision. Where the staff cells increase in number on several daily examinations and reach 12 per cent or more, operation is indicated. On the other hand, where the percentage of staff forms remain stationary below 12 per cent or shows a gradual reduction, operation can be postponed with safety and the patient kept under further observation." Such a conclusion as to the value of the hemogram is for the greater part agreed upon by other observers. Hesse¹¹ is in accord with the observa-

tions of Weiss,² Levy¹² and Heidemann,¹³ that this method gives more information than the total leucocyte count alone and bears out the belief that the blood picture is more valuable as a prognostic than as a diagnostic aid. While the author found the total W. B. C. count markedly increased in acute suppurative otitis media and mastoiditis, he was unable by means of the blood picture to differentiate the two and concluded that the most valuable aid given by the hemogram is in the recognition of major complications. Differentiation of complications is not possible by this method. Kaiser¹⁴ studied eighteen cases and found that grave complications, such as meningitis, brain abscess, sinus thrombosis and extradural abscesses, always manifest themselves in some form in the blood picture—either by a greatly increased W. B. C. count, high neutrophilia or a left sided displacement but that the latter was not invariable. He attaches great importance to the blood picture in determining whether the suppuration has been controlled after operation.

Here it is often a better index of latent abscesses and extension of a thrombus than are the fever and pulse. Gale¹⁵ cites the work of Adolph Glasscheib, in the January (1927) number of *Wien. Monatschrift für Ohrenheilkunde*, who makes use of an index based upon the relation of the unsegmented neutrophiles (juveniles and stabs) to the segmented (mature) neutrophiles—a count of 10 immature and 50 mature neutrophiles indicating an index ratio of 1:5. In cases of mastoiditis with a normal eosinophilia and an index of 1-10 to 1-8 no free pus and only granulations were found at operation; cases with an aneosinophilia and index of 1:3 showed free pus and granulations in the mastoid, while cases of perisinus abscess or sinus thrombosis had an index of 1-3 to 1-1 with a variable eosinophilia.

In 250 case studies in which the index was obtained, 40 per cent of the cases showed a distinct blood shift to the left. The index varied from 1:3.5 to 1:22. In 16 cases of purulent mastoiditis 20 per cent had normal or only slightly elevated W. B. C. counts with indices of 1:10 and upwards; 60 per cent had W. B. C. counts varying from 8,500 to 11,000 with indices of 1:4 up and 20 per cent with counts between 11-14,000 had indices from 1:5 up.

The author stresses the fact that the advent of a clinical mastoiditis does not disclose itself by marked changes in the blood, and the cases were operated upon because of clinical findings with very little help from the blood picture. Paradoxically, the lowest indices did not indicate the most severely ill. It was observed that the most marked and consistent blood changes occurred very early in the process—before rupture of the drum. The advent of a clinical mastoiditis, because of its gradual encroachment upon the mastoid cells and limited absorption via blood and lymph vessels, is not heralded by marked blood changes—the peculiar slow drainage from the mastoid allowing physiologic adaptation of the hematopoietic system to take place. In view of this fact, the lower the index (the higher the stab percentage) is in proportion to the length of the illness, the more severe the infection is apt to be. A high index, however, does not negate mastoid destruction, this being especially true in streptococcus mucosus mastoiditis. In the six cases of lateral sinus thrombosis and perisinus abscess there was 100 per cent shift of the blood with indices 1:1 to 1:5 and high W. B. C. counts (above 10,000). No differential diagnosis, on the basis of blood picture alone could be made between the two. He concludes that the index per se is unreliable in making a diagnosis between purulent mastoiditis, perisinus abscess and sinus thrombosis; also that the degree of blood shifting to the left has little value in determining the time of operative interference.

Levy¹² and Judd¹⁰ emphasize the importance of the rôle of the eosinophiles in certain conditions. Judd believes that the relatively extensive mucous surface made by the peculiar mastoid structure offers a large site for the absorption of the toxic substances into the blood stream. He considers that the invasive organism first sets up a septic process in the mastoid structure and is evidenced by leucocytosis, hyperneutrophilia and hypoeosinophilia. As the process continues, necrosis of the spongy cell partitions sets free the products of necrosis, now an alien protein, which, absorbed from the considerable area, is sufficient to evoke the appearance in the blood stream of the lytic agents which are those of leucocytosis, hyperneutrophilia and normal or

hypereosinophilia. The author concludes that the optimum time for mastoidectomy corresponds to the appearance of the lytic factors in the blood picture, which is interpolated between a mild preceding and an intense septic factor. Levy urges that a normal or changed blood picture should not deter one if operative interference is clinically indicated. He further emphasizes the necessity of eliminating such conditions as parasites, vagus irritation, certain skin diseases, allergy, scarlet fever and gonorrhea before the percentage of eosinophiles can be accurately evaluated.

The study of hemograms has been almost wholly based upon adult manifestations. Schilling repeatedly emphasizes this, and while he considers the reaction manifested in the blood after six years of age as approaching that of the adult, the greater viability of the bone marrow and variability of the lymphatic system response in children must always be considered. In the cases here reported and in others recently observed it has been found that while the hematopoietic responses in a few cases were difficult to interpret, the majority reacted similar to the conditions found to be present in the adult blood pictures. Ockel, Schilling and others also found that the hemogram findings were much more consistent in pathologic processes than when under apparently normal conditions. All of the cases observed in this reported series were in children varying from 5 to 13 years of age. Considerable similar work must be done before the hemogram in smaller children can be accurately interpreted.

CONCLUSIONS.

These observations have been limited in number, yet each case has been intensively studied, both clinically and hematologically. It is felt that the consistent manner in which the hemogram findings have paralleled the clinical features makes this laboratory aid of distinct value in the study of the otologic infections. Further confirmation is given in the fact that the stab cell count offers a much more reliable index of the patient's condition than does the polymorphonuclear percentage of the total number of the white blood cells, and particularly in children, than the pulse and temperature variations. The hemogram must be considered as

a distinct advance in the interpretation of various blood findings but cannot be expected to accurately differentiate between such closely allied conditions as acute suppurative otitis media and acute suppurative mastoiditis. It is also of far more value when daily or very frequent observations can be made under a strict set of conditions. It is by no means introduced to supplant clinical investigation but to corroborate it. Finally, the correct valuation of such laboratory criteria permits an individualization of treatment according to the given case, because it furnishes data so significant as to make pathologic details more comprehensible and the results more uniformly successful.

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XVII.

THE NOISES OF CIVILIZATION AND THEIR EVIL EFFECTS.

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Man many thousands of years ago became differentiated from the other animals about him by his utilization of tools.

Very crude they were in the beginning, but they enabled him to overcome his enemies, to secure food and live with a little less effort; the first step in the course toward civilization. It was by tools, therefore, that he began, figuratively as well as literally, to make some noise in the world.

Although it must have been evident that it was by implements and machinery that man extended the power of his arm and gained mastery over Nature, it seems strange to us now, in this age of machine civilization, that so many centuries passed with so little improvement in this regard. Each generation seemed perfectly content with what it had inherited from the generation preceding. It was really not until the latter part of the eighteenth century that the world seemed to awake to the idea that the forces of nature might be turned to the practical uses of man.

Then began that feverish search into her mysteries which resulted in many wonderful discoveries. Invention quickly followed discovery, and when it was seen that labor saving devices brought to the owner wealth and power, there soon sprang into existence a great multiplicity of machines.

Water power was first used, but this soon gave way to the more efficient power of steam, and then later was added the wonder-working power of electricity. Steam railways began to traverse the land; steamships to plow through the waters of the rivers, lakes and seas. The cotton gin and the spinning jenny were invented; sewing machines appeared in every household; harvesting machines on every farm.

A new age, the age of machinery, came into existence. A new era of civilization was proclaimed throughout the world.

Now every machine with its improved methods of production, and every invention turned to use for the manufacture of goods, or the transportation of man and material, brought also into existence more and worse noises.

In the excitement of the new-gained wealth and power of the dazzling new civilization, little heed was paid to the pernicious noises which accompanied.

It is significant, however, that it was about this time we first began to hear much of actual suffering of man from such a cause. We need only to mention as conspicuous examples the names of Schiller, Goethe, Carlisle, Dickens and Herbert Spencer, illustrious geniuses of that era, whose lives were made miserable on account of noise. With the profits and conveniences of the new industrial civilization came new afflictions chargeable to the same account.

In the early and simpler world, when man lived chiefly by the pursuit of agriculture, the sounds of the world were almost only those which emanated from animate and inanimate nature.

It has always been maintained and we believe rightly that the sounds of inanimate nature are good for the body and refreshing to the soul.

However unpoetical the mind, one can hardly fail to experience pleasure in the sounds of rustling leaves or of flowing water.

There is no doubt that much of the charm of music is derived from the conscious or unconscious suggestion of these elemental sounds, and we find therein the best explanation of why music soothes and exhilarates the spirit.

In strong contrast to the pleasing sounds of inanimate nature, are the disagreeable sounds of artificial origin, which we call noise—and whose effect is to ruffle, irritate and annoy.

Scientifically considered, music is characterized by a succession of regular rhythmical vibrations; noise by a medley of vibrations without order and uniformity. The contrast may be strikingly shown by graphic tracings. The musical note is represented by

regularly recurring perfectly formed curves; noise by crooked, irregular, crumpled lines.

The noise of the world may be roughly classified as originating from the following causes: 1, animate nature; 2, war; 3, building and construction; 4, traffic and transportation; 5, manufacture; 6, commerce; 7, communication.

Our forefathers who tilled the soil, hunted wild and took care of domesticated animals, were not altogether free from the annoyance of noises.

The sounds emitted by animals are in the main for two purposes, to attract mates and to terrify enemies. The former are intended to be pleasing, and in truth we do not as a rule find disagreeable the cooing of doves, the neighing of horses, the mooing of cows. The warbling note of the song bird gives almost universal delight.

But we may class as noises the sounds of animals made to terrify and drive away foes. The roar of the lion and growl of the tiger must certainly be so considered.

Much, however, depends upon the time and place. The distant bark of a dog is sometimes good to hear, but coming from the back yard in the early morning hours it is an unpleasant noise.

War, since the earliest times,* has been a prolific source of noise. The warriors of old were spared the terrific din of heavy artillery, but they managed somehow to make enough noise to "fright the souls of fearful adversaries." The noises of modern warfare are stupendous and overwhelming and their harmful effects fall upon friend more than upon foe.

The noises incident to construction are probably the most intense of all those to which the modern city dweller is exposed.

It is said that the erection of the great steel skyscraper takes a heavy toll from among the workmen because of the extra hazard-

*A graphic account is to be found in scripture of the successful employment of noise in war for the purposes of "*Schrecklichkeit*." "For the Lord made the host of Syrians to hear a great noise of chariots and a noise of horses and they said to one another, Lo the King of Israel hath hired against us the Kings of the Hittites and the Kings of the Egyptians to come upon us; wherefore they arose and fled in the twilight and they left their tents and their horses and their asses, even the camp, as it was and fled for their life."—II Kings, 7:6.

ous nature of some of the work. There ought also to be charged to it certain ills of which no reckoning is usually made, and which affect not only those engaged in the work, but also the innocent neighbor and the disinterested bypasser.

There is nothing that better bespeaks the wisdom of Solomon than the precaution he took for the noiseless construction of his famous temple.

As his own pastoral people were unskilled in such work he engaged artisans from Tyre and Sidon, but he directed that all the work of cutting and chiseling of the stone should be done a distance away so that, as it is written, "no sound of hammer or ax or any tool was heard in the house while it was building."

Would that we had a Solomon to protect our people from the ruthless builders in our great cities. He would save many from ruined ears and shattered nerves. It is not now a matter of such mild noise as that of hand-plied hammer and ax and saw—but the unearthly din of rock drills, excavating shovels, pneumatic hammers and, worst of all, riveting machines.

Scientific measurements have been made throughout the city of New York of noises from different sources, and the results recorded in terms of the decibel.

The noise of riveting measured at close range registered 100 decibels, which means that the intensity of this sound was ten billion times the minimum sound that can be heard.

In the survey of New York City noises made by the Noise Abatement Committee especially appointed to study the question and report on these matters, it was found that 36 per cent of the noise was chargeable to traffic and 16 per cent to transportation. As a matter of fact, it seems impossible to separate these items.

Vehicular traffic is now almost altogether a matter of automobiles and naturally with over twenty million of them in this country with their horns, sirens, whistles and bells constantly on the go they are a prolific source of noise.

The worst offenders are the heavy busses, vans and trucks, especially when running on solid tires, or with loose gears and chains.

The noisiness of city streets is tremendously increased by the public carriers, the surface trains, the elevated and the subway.

The clatter and clang of street cars are harmful as well as disturbing to those who have to live along these routes, and conversation and business have often to be suspended at the moment of their passing.

The noises of the subway and of the elevated are of even greater intensity at close reach and they contribute much to the general street noise.

According to Dr. E. E. Free, the noisiest corner in the world is 34th Street at 6th Avenue, New York, a location, as he remarks, cursed with three main streams of street traffic, three surface car lines and two tracks of the elevated railway.

Here the intensity of the average noise is 100,000 that needed for hearing.

Dr. Harvey Fletcher, in his thorough, painstaking measurement of New York City noise, found corners in which at times even this noise is exceeded.

The noise of steam railway transportation has in recent years been improved somewhat by better roadbeds and better built cars. The noise of freight cars, especially when backing and filling on a side switch, with the coupling and shrill whistles of engines, leaves, however, much to be desired.

The noises of water transportation are in general not so bad as those of land but for persons who live near water fronts the foghorns and whistles of tug-boats and other craft is often very disturbing, especially at night.

The newest arrival in the field of transportation is the airplane. It has a noise all of its own, and usually of such intensity that passengers and pilot must wear earplugs and forego all conversation while en route—a noise of such stunning effect to some that they will be deafened for many hours after landing.

As noises coming under the head of commerce we would especially mention those connected with the collection and delivering of merchandise. The throwing about of boxes and barrels and tin containers are only a few that could be mentioned.

The early morning call of the iceman and the milkmen are particularly dreaded in some quarters.

We must also include here the weird call of hucksters, the hoarse shout of newsboys calling "Extra!" the bell-ringing of

scissors grinders—all noises with which most of us would willingly dispense.

The recent great increment in the noises of civilization is attributable to the coming into common use of measures for more rapid communication, the telegraph, the telephone, and the radio.

Of all the most notable is the radio. The click of the telegraph key or the bell-ringing of the telephone receiver seldom bothers anyone except those directly in the room, but the squeak and squawk of the radio are often heard through the partition walls of the apartment, and sometimes carried out of the windows to the neighbor across the street or people out on the sidewalks below.

Under this head also we must include the typewriter, an instrument of communication in very common use. Notwithstanding some decided improvements that have been made in this regard, most offices still cling to the old style hammer-blow type of instrument either by reason of habit or on the score of economy. When health is considered, however, it is poor economy. Indeed we doubt not that business economy would be better served by a quieter environment, for under the influence of distracting noise efficiency is everywhere reduced.

Errors are likely to creep into accounts, letters are formulated with more difficulty and conversations with clients may be less convincing.

We have not, of course, named all the troublesome noises there are, but enough to be sure to indicate that this present work-a-day existence of ours is indeed very full of noise.

The past decade or so has been marked by a very extraordinary advance in science and multiplication of machinery and it was inevitable that noise should correspondingly increase.

The most alarming fact is that noises have not only increased in number, variety and intensity but also in extension.

Formerly confined to certain special localities, as factories, railroad centers and cities in distinction to the country, now thanks to the automobile, the flying machine, the power plant and the radio, the dominion of noise has been enormously extended.

Indeed the noises of civilization now not only cover the face

of the earth but they fill the air above and even invade the water under the earth.

There was a time when the tired city dweller might easily find a sequestered nook in the country, where all was quiet, restful and serene, but where can one go now and not be obliged to hear raucous horns or screeching sirens or (if by the waterside) the eternal chug-chug of motor boats and their shrill whistles.

There is nothing more certain than that he will have a continual treat of filtered jazz music from the inevitable radio and always possible that he may have forced upon his ears the thunderous roar of low flying planes, which are no respectors of persons and are not restricted by the laws of eminent domain.

EVIL EFFECTS OF NOISE ON HEALTH OF MAN.

The evil effects of noise may fall chiefly on the auditory apparatus itself, or chiefly on the general nervous system.

The hearing organ may be affected in one of three ways: (1) by suffering loss of function; (2) by developing a state of abnormal sensitiveness, or (3) by acquiring a special tolerance or habituation to the noise irritant.

It is a fundamental physiological law that an organ treated to an excess of its proper stimulus must either adapt itself thereto or suffer harm.

The auditory sense, phylogenetically considered, is the most recent arrival in the family of special senses. It is therefore of frailer texture and endowed with a feebler resistance than any of the others.

This is no doubt the explanation of the well attested clinical observation that when the eighth nerve, composed equally of auditory and vestibular fibres, comes under the influence of toxic material in the blood, the former proves regularly to be the more vulnerable and it makes likewise understandable the fact that the auditory function can readily suffer from overstimulation by sound.

It is pertinent in this connection to call attention to the comparatively unprotected state of the hearing organ.

Its situation deep within the petrous portion of the temporal bone is indeed a good guarantee from ordinary gross trauma-

tisms, but we refer to the helpless exposure as regards the vibrations of sounds. The retina is protected by eyelids which close voluntarily or involuntarily against injurious visual stimuli, but there are no earlids to save Corti's organ either night or day from hurtful noises that fill the surrounding air. The tympanic muscles are perhaps intended to have a function somewhat analogous to ciliary muscles, but if so they are obviously inadequate, because of their feeble action and their easy susceptibility to fatigue.

Deafness is not so common a result, notwithstanding the frequently expressed opinion of those who are waging war against the noise nuisance. It can certainly occur when the individual is exposed to an intense noise, at close quarters over a long period. Such conditions sometimes present themselves in certain occupations, as the boilermakers' trade, where deafness is due to a nerve degeneration of high degree and incurable, and appears in a large proportion of cases.

It is a well founded observation, however, that such results occur only when the prevailing notes of the noise are in a high pitched register and of a very disagreeable character.

Some interesting experiments have been made by Witmaack, Yoshi, Siebenmann and others of exposing animals continuously in close proximity to intense sounds of various kinds and later examining postmortem the deafened ears. Degenerative changes were found in Corti's organ, and it is notable that they were chiefly located, in confirmation of Helmholtz's doctrine, in parts of the cochlea corresponding with the pitch employed.

Sometimes the middle rather than the internal ear is affected by excessive noise. Sudden intense explosives, such as those of bombs and heavy machine guns, may spend their force on the middle ear, causing rupture of the tympanic membrane and hemorrhage. This may act as a safeguard against injury to the labyrinth, but labyrinthine concussion may nevertheless at the same time occur.

Now, the noises to which the average citizen is exposed are seldom of such nature and intensity as to produce deafness. The usual city noise, for instance, is that of a continuous roar and hum, with only occasional severe exacerbations.

What happens to the individual much exposed in this way is one of two things, either he develops for the noise excessive sensitiveness or he develops an especial tolerance—auditory hyperesthesia, or the noise habit.

Auditory hyperesthesia is a much more common affliction than from the literature of the subject we would be led to believe.

If the otologist would more often follow the clue given by the patient who casually mentions that shrill sounds produce on his ear a decidedly painful impression and that even ordinary sounds are distorted into unpleasant, clangy effects, the diagnosis would be made oftener, I am sure, than it is.

Annoying tinnitus is also a frequent symptom, and in some cases even static functions are affected, as evidenced by more or less vertigo.

These cases uncomplicated by a middle ear disease require, of course, not local treatment but a prolonged rest in a quiet country place if such can be found. When obliged to remain in the same environment much help can be obtained by keeping the ears continually plugged by cotton or wool moistened with glycerin.

Middle ear inflammation may be present at the same time, associated with hyperemia, which tends to exaggerate the sensitiveness. In such cases, of course, the middle ear affection should have appropriate treatment, which is often found to be quite helpful.

Noise Habituation.—Since it is exceptional for the ordinary noise of our environment to be of sufficient severity to produce deafness, adaptation may take place by the development of a certain tolerance for the customary noise, which eventually may take on the form of a regular noise habit.

There is no question that many persons do become so habituated to noise and so dependent upon it that they seem unable to get along without it. It is not an uncommon experience, as most of us very well know, for persons who have lived a long time in the midst of city noise to find, on suddenly being translated to the quiet of the country, that the silence is actually oppressive.

They are unable to sleep and are restless and unhappy until back again where they can hear the noise of the city streets.

There are many persons of whom it may be said that they develop an actual fondness for noise a *ptupophilia*, and so are constantly seeking pleasure in noisy excitation.

In some cases the tendency goes on to the extent of a craze or mad craving for noise, a veritable *ptupomania*.

This is a malady which, we fear, has become already alarmingly common in America and which we believe explains the widespread popularity of the degenerate jazz.

Disorders of the Brain and Nervous System.—The injurious effects of sounds are in many instances not confined to the ear but pass over to the brain and general nervous system.

Sounds affect us through the association of ideas. The soothing or soul-stirring influence of music is explicable, as we have already intimated, by the conscious or unconscious suggestion of the sounds of inanimate nature.

Noise, on the other hand, may be hurtful by the suggestion of physical harm and suffering.

Drawing the finger nails over a rough surface or biting on a gritty substance is accompanied by sounds peculiar to these acts. Such a sound when reproduced naturally suggests injury to the human organism and gives a disagreeable impression. There is recognition by inference of association of ideas when certain noises are spoken of as harsh or grating, and even more directly when described as having the effect of "making the blood run cold" or causing "cold chills to run up and down the spine."

Another reason to account for the disagreeable effects of noise upon the nervous system is the arousing of what is called the fear-reaction, an instinctive reaction inherited from our remote ancestors, who had to be always on guard against surrounding enemies and to whom certain menacing sounds were a signal of impending danger.

This applies especially to sharp, sudden sounds, which break unexpectedly upon an otherwise comparatively quiet environment. We know how the effect of such sound is to make us start.

Because of the importance of the association of ideas, we can understand that the nervous system is concerned not so much with

the intensity of the sound,—the number of decibels that it registers, as with the disagreeable character and the suddenness.

A number of experiments have been made which show that noises have a decided effect on the vital functions of man and the lower animals.

Couty and Charpentier found that noises, such as the slamming of a door, caused in dogs a 10 per cent rise of cardiac tension with acceleration of the pulse; and Landis observed in man a rise of systolic pressure of 20 m.m. as a result of the explosion of a firecracker.

Particularly interesting were the experiments reported by Dr. Foster Kennedy on cases in the hospital which, on account of accident or operation, permitted observation of direct effect of sound on brain circulation. It was found that explosion of blown up paper bags caused a notable rise in intracranial pressure. Experiments have been made by Dr. J. B. Morgan of North Western University, by Dr. A. T. Poffeberger of Columbia University and by Donald A. Laird of Colgate University to determine, especially from the psychologic point of view, the effect of intense noises on mental functions and incidentally its influence on other body functions.

In practically all these experiments there was a speeding up of the motor, cardiovascular and respiratory functions; and in the performance of various mental operations there was an undue strain and fatigue and loss of energy in comparison with silent conditions.

Such experiments form a basis for the explanation of the neurasthenias and psyasthenias which are reported by authority of clinical experience.

It is worth mentioning that from various parts of the country recently reports have come which indicate an unfavorable effect of loud noises on the reproductive functions. One farmer reported that because of the nearness of an aerodrome and the terrific noises, the cows on his farm failed to calve and gave less milk; another threatened to bring suit against a company because the egg-laying of his hens had been reduced to nil.

I know of no experiments directly to test the effect of sound on the reproductive function, though Dr. Laird has observed that

the growth and development of rabbits in noisy cubicles was decidedly poor, as compared to the control, and he is of the opinion that severe noise may unfavorably affect the nutrition of infants.

It may or may not be significant that there has been a steady decline of the birth rate in all civilized countries which dates from the beginning of the machine age.

In England and Wales the birth rate in 1877 was 36.2 to every thousand of the population; in 1928 it had fallen to 16.7. From similar high rates it fell in the United States to 18.2, and in France to 18.2, Germany to 18.6 per 1000. In other countries where machine civilization is not a factor the rates have remained high; in Ceylon, for instance, in 1928 it was 40; in Egypt 43.3 per thousand.

Of much greater importance than the falling birth rate is the rising incidence of mental diseases, which has been noted in all civilized countries in recent years. This has gone on until at the present time, in the United States for example, we are confronted by the appalling reality that the inmates in the institutions for the insane and feeble-minded outnumber those hospitalized for all other disabilities combined.

The significance of this increase in relation to noise cannot be ignored, because of the experimental proof on the one hand of the influence of noise upon brain function and on the other the clinical testimony to the fact that mental derangement is often directly traceable to this cause.

One factor, as already mentioned, is the strain which induces mental fatigue and irritability; another, no doubt, the loss of sleep and rest which have ever been recognized as potent causes of mental breakdown.

Consideration of these factors, pointing to the detrimental influence of noise upon intellectual faculties, brings us face to face with the question whether or not noise, the inevitable accompaniment of the higher civilization, should not be accounted civilization's worst enemy.

Advance in science is made possible only through sustained thought and concentrated attention. Noise scatters thought and is an effective hindrance to the operation of attention. Rest and sleep are fundamental necessities for the continuance of healthy

mental activities. Noise produces fatigue and irritability and then denies the sleep which is more than ever needed to restore mental function.

It is then an inescapable conclusion that noise, the by-product of civilization, unerringly tends to impede and destroy those very intellectual functions upon which progress of civilization depends. It is the ash which unremoved will eventually extinguish the fire.

The very discouraging thing about this positive evil which we call noise is that, bad as it is, the future looks worse.

Edison, referring to the future of the cities, says that city noise must inevitably grow greater, and that the man of the future generation will as a consequence be deaf. Without accepting the pessimistic doctrine that the ultimate destiny of the city dweller is deafness, we have no doubt that the noises of the civilized world are steadily on the increase, for not only are daily new machines being made, with a consequential increment of incidental noises, but—and this is the crux of the matter—machines are being now devised and coming into use which make the increase or the magnification of sound their main or primary purpose.

The most ingenious perhaps of modern inventions are those which have to do with the transmission and amplification of sound. Fortunately for us, the sounds of our hearts are not ordinarily heard and the contraction of the innumerable muscles of our body take place in silence.

But it is now possible by means of a little disc held in contact with the body to cause the pulsations of the heart to be heard throughout the room as the thumping of a heavy hammer, and the contraction of minute invisible muscle cells, 1/5000 of an inch in breadth, to be audible as loud crackling explosions. Is it not awe inspiring and perhaps ominous for the future, to meditate upon the possibilities if all the silent world were wakened into sound?

A short while ago, Pastor Wagner, author of the "Simple Life," lamenting the noise and bustle of the age, found consolation in the fact that "after all, the realm of silence is vaster than the realm of sound." So perhaps it is, but there is reason to fear that it cannot always remain so, unless the efforts of science to

suppress noise can in the future be made to keep step with efforts to create it.

There is need for arousing public interest in this direction, for notwithstanding the activities of a committee here and there, marked indifference on the subject generally prevails. Let a new wonder making machine come to light, and you find always the interest in its accomplishments easily blinds us to the evils of its noise.

The need perhaps is more acute because of the increasing number of ptupophiliacs among us, who love noise, not for what it means but simply for noise sake.

For esthetic reasons alone a strong sentiment has been worked up to rid the highways of billboards and ugly signs which offend the sight.

Shall we be less active to get rid of noise which not merely offends the hearing but injures it and which may do harm to the health of the body and mind, and which, furthermore, there is good reason to believe hinders the future upward progress of the race?

The word civilization is said to be derived from the word "quies," meaning rest or quiet; the idea being that through the freedom from the necessity of labor one secures leisure, rest and quiet; but the unexpected has happened and there has come instead unrest, disturbance and noise.

Will it always be so? Or will not science find some effectual means to rid us of this unwholesome by-product, so that civilization may eventually reach the goal which is its aim?

XVIII.

THE SUPPURATION OF THE PETROUS PYRAMID:
PATHOLOGY, SYMPTOMATOLOGY AND
SURGICAL TREATMENT.*

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PART II.

SYMPTOMATOLOGY.

The symptomatology of suppurations of the petrous tip can be divided into four periods: The period of the eye pain and aural discharge; the period of low grade sepsis; the period of quiescence; and the terminal stage. As a rule, only the fourth stage appears as an entity; but in order to develop the clinical picture so as to describe a sequence of events, the grouping of the symptoms as described is deemed advisable. Later on, the entire symptomatology will be correlated. In this section we propose to explain each individual symptom, give its clinical significance and analyze its anatomic and pathologic cause.

The Period of the Eye Pain and Aural Discharge. Preliminary Data.—All of our cases occurred in patients whose mastoid processes showed extensive pneumatization. Before the primary operation on the mastoid process, extensive ramification of the cellular elements was always noted on radiographic examination. At operation, this observation was verified. The cells themselves,

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or the spaces created by their coalescence due to the disease, were found to occupy large areas of the zygoma, squama and occipital bone and to surround the base of the petrosa completely. The region of the so-called solid angle contained cellular elements or showed areas of necrosis. These findings are significant for, by demonstrating that the process of pneumatization has extended beyond the limits of the mastoid process, they suggest the possibility that the petrous pyramid is pneumatic. The presence of extensive pneumatization in the temporal bone should help one to interpret later signs and symptoms, if and when they appear.

Eye Pain.—This is, in the majority of instances, the first symptom to make its appearance. Its location and character are so typical that it is almost diagnostic of petrosal tip suppuration in itself where the anatomic structure already described has been found.

The pain is on the side of the lesion. It is limited to the region about the eye and is felt within the orbit itself. It is described as a deep seated ocular pain and, at the onset, is nocturnal in character. During the day the patient is more or less comfortable; but, as the evening comes on, the pain becomes more and more intense. The patient describes it as being "just above the eye and through the eyeball."

This peculiar type of pain is highly significant of a petrosal tip suppuration. It is the result of an irritation of the ophthalmic branch of the trigeminal nerve, which, as has been pointed out in the first section of this paper, is firmly bound down in its course from the ganglion to and through the cavernous sinus. This branch, altogether sensory in function, supplies the eyeball, the lacrimal gland and the skin of the nose, upper eyelid, forehead and scalp.

The presence of retro-orbital pain early in the disease is explained by Eagleton¹³ as due to an inflammatory reaction of the dura overlying the diseased petrous tip and the petrosphenoid articulation. Ostmann²³ stressed the significance of supra- and infraorbital neuralgia and deep seated eye pain in his case. Streit⁴³ also noted eye pain on the side of the lesion. In an

analysis of the clinical picture presented by his two cases, Lange⁹ gave prominence to the deep seated eye pain.

In our cases, eight presented this typical eye pain as the initial symptom. Table 1 shows the time of appearance of this symptom in relationship to the onset of the otitic disease and the primary operation. It will be noted that in one case (case 4) the pain was present from the onset and was not relieved by any of the surgical procedures instituted prior to the drainage of the petrosal tip. In the remaining cases, a varying interval of time elapsed between the performance of the simple mastoidectomy and the beginning of the eye pain.

TABLE I.

Case No.	Location of Pain	Time of Onset	
		Re: Otitis	Re: Operation
1.	Right frontal headache	45 days	18 days
2.	Pain through left eye	54 days	45 days
3.	Pain through right eye	7 days	Constant
4.	Pain through left eye	42 days	12 days
5.	Pain through right eye	108 days	27 days
6.	Pain through right eye and temple	34 days	20 days
7.	Pain through left eye	40 days	20 days
8.	Pain through right eye	60 days	25 days
9.	Pain through left eye and over eye	16 months	*7 months

*After third operation.

The presence of postoperative pain after surgery on the mastoid process is to be expected. Where, however, this pain assumes definite characteristics, it is usually significant of some complication. A dull, aching pain felt on the side of the head and persisting for a time after operative interferences is very significant of lateral sinus thrombosis (Phillips⁴⁴). Pain in the nape of the neck or in the occiput makes one suspicious of an impending meningitis if it follows mastoidectomy. Similarly, deep seated eye pain is a distinct symptom of a lesion in the middle cranial fossa which is irritating either the ophthalmic branch directly or its root in the semilunar ganglion. A posterior fossa lesion cannot, for anatomic reasons, involve the region of the Gasserian ganglion. The tentorium so completely separates the middle from the posterior fossa that a localized purulent focus within the

latter cannot possibly, even with its surrounding inflammatory reaction, involve an isolated portion of the fifth nerve, which is situated wholly within the middle cranial fossa.

Other branches of the fifth nerve besides the first may be involved if the inflammatory reaction is sufficiently widespread. Pain will then be felt all along the area supplied by the second and third branches. This pain is not diagnostic, however, as it can be associated with cases of uncomplicated middle ear abscess and mastoiditis. Any irritation of the geniculate ganglion of the facial nerve may cause pain to be referred to the area supplied by the superior maxillary branch of the trigeminus. This occurs through the communication established between the geniculate ganglion and the second trigeminal branch of the great superficial petrosal nerve. The mandibular branch of the fifth, through its connection with the otic ganglion, receives sensory fibers from the small superficial petrosal nerve. This may be irritated either at its origin in the tympanic plexus or in its passage through the petrosa to reach the otic ganglion. Irritation of Jacobson's nerve or the tympanic plexus may cause pain to be referred along the distribution of the superior maxillary nerve through the great deep petrosal, which joins with the great superficial petrosal.

It is therefore clear that pain in the face and teeth can occur with a suppurative lesion located anywhere in the middle ear or mastoid process. This pain will be relieved, however, as soon as the source of irritation in the middle ear or mastoid is removed. Pain felt in the region of the orbit, due to irritation of the ophthalmic branch of the fifth, must be caused by a lesion in direct proximity to this branch, for it has no connection with the other sensory nerves in the petrosa.

When surgical removal of the purulent focus in the mastoid process and middle ear does not result in a cessation of the pain distributed over the areas supplied by the second and third branches of the fifth nerve, the persistence of the pain should be viewed as suspicious of a petrosal tip suppuration, when it is continuous in nature and not of the spasmodic type. Spasmodic pain is more apt to be associated with an idiopathic lesion of the Gasserian ganglion itself, like tic douloureux or neuritis. In

petrosal tip suppuration we are more likely to get a constant ache than a spasmodic pain.

The Aural Discharge.—It has been the experience of most otologists that a simple mastoidectomy which has established adequate postauricular drainage will cause the middle ear to cease discharging within a week or two after operation. In our cases of petrosal tip suppuration, either the middle ear continued to discharge until the lesion in the petrous tip was identified and eradicated, or else, after a period during which the ear was dry, a profuse discharge suddenly reappeared, at the same time as, or shortly before, the onset of the eye pain.

In the first section of this paper we have discussed the significance of petrosal tip suppurations as the cause of a type of chronic otorrhea in patients showing a well pneumatized mastoid process. An acute middle ear suppuration which has a coalescent mastoiditis as a complication will, in most cases, undergo resolution after the mastoiditis has been eradicated. The otorrhea may persist after simple mastoidectomy on a well pneumatized mastoid process if the acute lesion in the middle ear was of the type known as the "acute necrotic otitis."³⁹ This variety occurs in scarlet fever and in the course of a debilitating disease such as diabetes. When it is present, the greater portion of the mucosa of the tympanum undergoes necrosis, exposing the underlying periosteum and bone to the suppurative focus. A rarefying otitis, or caries, results which is evidenced clinically by a marginal perforation and bare bone. In the reparative stage of the lesion the squamous epithelium of the external auditory canal plays a part in the attempted cure of the pathologic state, and an ingrowth of epidermis occurs which eventually results in a secondary cholesteatoma. Every otologist is familiar with the rapid appearance of a cholesteatoma in certain cases of this type wherein a thorough simple mastoidectomy has been performed. The squamous epithelium, in its ingrowth into the middle ear, finds a large preformed cavity in the mastoid process, into which it grows rapidly, eventually forming a large cholesteatoma. This, then, is the other cause of a continued otorrhea following acute suppurative lesions in the presence of a well pneumatized mastoid process.

TABLE II.
PERIOD DURING WHICH EAR WAS DRY AFTER SIMPLE
MASTOID OPERATION.

Case No.	Dry	Onset of Discharge in Relation to Eye Pain
1.	5 days	6 days prior to pain
2.	19 days	14 days prior to pain
3.	Never dry	
4.	7 days	Day of pain
5.	6 days	5 days after pain
6.	6 days	1 day prior to pain
7.	Never dry	
8.	10 days	1 day after pain
9.	7 months	1 week after pain

In our cases of petrosal tip suppuration, the continued otorrhea could not be accounted for by any of the foregoing factors. Neither the otoscopic picture nor the pathologic findings at operation agreed with that presented by cases of bone caries or secondary cholesteatoma. None of our cases showed a marginal perforation at any time. In seven cases the drum had completely healed, and the aural discharge recurred only after an interval of time (Table 2). At the second operation nothing could be seen in the previously exenterated mastoid process to explain the recurrence of the otorrhea as the result of a reinfection or a faulty performance of the simple mastoidectomy. In one case (Case 7) we performed a revision of the simple mastoidectomy, fearing that we had overlooked a suppurative focus at the primary operation; but we found a healthy cavity, completely filled in with connective tissue undergoing ossification at its periphery.

In the performance of the radical operation on our cases, however, we were impressed with the amount of granulation tissue present in the antrum or middle ear. In seven cases it was situated around the tubal orifice. The tubal mucosa was markedly edematous, whereas that covering the promontory and fallopian canal was glistening and thin. No areas of necrotic bone were present within the middle ear. In the remaining two cases a large fistula was found above the horizontal and behind the superior semicircular canal, leading inward and forward toward the tip of the pyramid. The opening of this fistula was surrounded

by granulations, and mucopus exuded from it. The middle ear was normal.

Uffenorde²⁶ considers the reappearance of otorrhea in conjunction with eye pain a significant factor in the symptomatology of petrosal tip suppuration. He states when an acute middle ear suppuration is protracted after operation, one must think of a suppuration of the pyramidal tip. Eagleton's¹³ cases, as well as others reported in the literature, all show this recurrence or continuance of a discharge from the tympanic cavity.

As already stated, we explain the recurrence of the otorrhea on the basis of the observations made by Lange⁹. In most instances, the pus in the petrous tip drains into the middle ear through the channel created by the peritubal cells. In some cases—in two of ours—the pus drains out through the avenue of invasion, which is located in the region of the inner antral or inner epitympanic wall.

Other Symptoms and Signs Occurring During this Period: It has been our experience that the eye pain and the aural discharge constitute the salient findings which are invariably present early in the course of a petrosal tip suppuration. There have been other signs which occasionally present themselves in the early stage of the lesion. They are included at this point merely as corroborative, but not as diagnostic data.

Facial Weakness: Two of our cases showed a distinct interference with the function of the seventh nerve early in the course of the disease. In one case (Case 2) this symptom appeared prior to the onset of the eye pain. In the other (Case 7) its appearance coincided with that of the eye pain. The disturbance in function was of the infranuclear type; at no time was there a complete loss of facial movement on the affected side. The duration of the facial weakness was short. In Case 2 it lasted four days. In Case 7 it lasted six days. The facial palsy can therefore be described as transient in nature; it occurred in the course of the disease but cleared up as the disease progressed.

The facial nerve is affected in the spread of the infection from the inner antral wall through the perilabyrinthine cells. Where this tract of cells runs in a retrolabyrinthine direction, that is,

behind the superior semicircular canal, the infection will not encounter the facial nerve in its passage through the petrous bone. Where the infection spreads into the pyramidal tip through the peritubal cells, the facial will not be involved at an early date. It is only where the tract of cells encounters the facial canal in its passage through the petrosa, that an infection traveling along this tract may involve the facial nerve. A transient paralysis, occurring in the postoperative period of an acute mastoiditis and accompanied by deep-seated eye pain and profuse aural discharge, is therefore significant of the advance of an infection into the petrous tip through the tract of cells leading from the mesial wall of the antrum.

In the two cases mentioned above, the facial paralysis was accompanied by vertigo and nystagmus.

Vertigo and Nystagmus: These are also produced by the perilyabyrinthitis and are also transient in nature. That they are not the result of a lesion located within the perilymphatic or endolymphatic space, can be readily determined by functional examination of the labyrinth. This elicits normal responses to both rotary and caloric stimulation. The fistula test was negative in our cases and the cochlea perceived sound stimuli. The Weber test showed a lateralization of sound to the diseased side.

The vertigo was not in any particular direction; the patients merely stated that they were dizzy. Neither did the nystagmus have a distinct character or direction. Sometimes it shifted in direction within a few hours. None of the patients assumed the forced position seen in cases of suppurative labyrinthitis. The vertigo and nystagmus disappeared in both cases at the same time as the facial palsy did.

Vomiting: Some of our patients had occasional vomiting spells during this period of the disease. The vomiting was not projectile and did not have any definite relationship to meals. The patients or their parents attributed it to "car sickness" or a "spoiled stomach." It usually occurred only two or three times, and lasted only a day or two.

The Period of the Low Grade Sepsis: The temperature in our cases of petrosal tip suppuration was that of a low grade sepsis. For a detailed study of the febrile reaction, see Table 3. On an

average the temperature was low in the morning, between 99 and 100. Toward the late afternoon, it would rise to 101-102.

Postoperative temperature following mastoidectomy may have many causes. Consequently, no significance can be attached to it unless other symptoms are also present to help identify the source of the fever. When a low grade sepsis continues after mastoidectomy, accompanied by eye pain and aural discharge, it is to be viewed as very strong corroborative evidence of a petrosal tip suppuration. The presence of fever indicates an inflammatory disease; the presence of eye pain indicates irritation of the ophthalmic nerve. The two together indicate an inflammatory lesion in the vicinity of the ophthalmic nerve.

TABLE III.
TEMPERATURE RANGE

Case No.	1st, 2nd and 3rd Periods		Terminal Period	
	A.M.	P.M.	A.M.	P.M.
1.	100.0°	103.0°		Recovered
2.	100.2°	102.0°	101.0°	106.0°
3.	99.0°	102.0°		Recovered
4.	99.8°	102.5°	102.0°	105°-106°
5.	99.0°	102.0°	100.0°	105.6°
6.	99.0°	102.0°		Recovered
7.	99.6°	101.2°		Recovered
8.	100.0°	103.0°	102.0°	106.0°
9.	99.4°	100.5°		Recovered

It may be said that the aural suppuration in itself can produce fever. This is true; but with the additional factors of extensive pneumatization, the eye pain and perhaps vertigo and nystagmus, the fever assumes an added significance and points to the diagnosis of a suppurative focus within the apex of the petrous pyramid. Eagleton considers the low grade sepsis an equally important diagnostic factor as the eye pain. He says, "Pain in the first branch (of the fifth nerve), limited to the region behind the eye, is significant of irritation of the dura over the petrous apex and, in the presence of continued sepsis, signifies caries of the petrous apex."¹³

We have found that the fever stays at a low level until a meningitis appears or until the pus in the tip has been drained. In the

former event a sharp rise in temperature is noted; and the fever then continues at the high level characteristic of meningitis. Where drainage has been established, either spontaneously or by operative measures, the fever subsides and the temperature gradually returns to normal.

The Period of Quiescence.—In most of our cases (see Table 4) there occurred an interval of freedom from all pain of diagnostic import. This period of quiescence varied in duration from five to nineteen days. Before proceeding further, it must be repeated that the pain to which we are referring is the deep seated eye pain, associated with a low grade sepsis. As previously shown, the presence of trigeminal neuralgia alone or of pain not limited to the first branch of the trigeminal nerve in no way serves as a diagnostic symptom of petrosal tip suppuration. Therefore, the presence and subsequent disappearance of pain in the areas supplied by the second and third trigeminal branches do not create what we designate as the period of quiescence. We refer only to the presence of deep seated eye pain, in the company of low grade sepsis, and to the subsequent abatement of this pain.

TABLE IV.
DURATION OF PERIOD OF QUIESCENCE

Case No.	Days
1.	2
2.	7
3.	2
4.	3
5.	6
6.	2
7.	None
8.	None
9.	None

From the standpoint of the patient's safety, this period is the most dangerous one, since it may lead both patient and surgeon to conclude that the lesion is clearing up. On the contrary, in the majority of cases, this period coincided with the invasion of the endocranium. In only one instance (Case 7) did it signify spontaneous evacuation of the purulency in the tip through the middle ear.

When it is understood, as we have previously pointed out, that the eye pain is due to traction exerted on the ophthalmic branch because of the inflammatory swelling of the dura in the region of the petrous tip, it will also be understood that the relief of this inflammation will result in a cessation of the pain. We are familiar with the fact that the pain in an acute coalescent mastoiditis is considerably lessened when the pus in the mastoid process ruptures through the cortex and forms a subperiosteal abscess. This is exactly what occurs in cases of petrosal tip suppuration. The localized collection of pus within the apex at first causes an edema of the overlying dura with a consequent traction of the ophthalmic nerve, which is in proximity to it. As the lesion progresses, if sufficient drainage is not established through the peritubal cells or through the tract of invasion, the upper surface of the apex becomes eroded, either directly under the Gasserian ganglion or through the thin bony partition which separates it from the carotid artery. These are the two likely places for erosion, as the posterior wall of the apex is anatomically much thicker than the upper and outer, and consequently offers a greater resistance to the carious process.

Once a perforation has been formed, the pus makes its way subdurally in the region of the Gasserian ganglion. There results an extradural abscess, which bears the same relationship to the disappearance of pain in the lesion under discussion as does the formation of a subperiosteal abscess in an acute coalescent mastoiditis: the inflammatory tension on the dura in one instance, and on the periosteum in the other, is alleviated.

The question now arises as to whether or not the direct involvement of the Gasserian ganglion by the purulent focus will result in the continuation or intensification of the pain. From the report of Turner and Reynolds⁴⁵ and that of Eagleton,⁴⁶ it is doubtful whether inflammatory infiltration of the Gasserian ganglion itself will produce pain. Turner's and Reynolds' case was one of cavernous sinus thrombosis wherein an inflammatory infiltration of the ophthalmic nerve was demonstrated postmortem and yet the patient had had no pain behind the eye. Eagleton's case 24, one of cavernous sinus thrombosis with purulent infiltration of the semilunar ganglion, had no trigeminal pain.

TABLE V.

OTHER SYMPTOMS FOUND DURING VARIOUS PERIODS.

Case	Cranial Nerves						Nystagmus	Vertigo	Vomit.
	6th	7th	8th	9th	10th	11th	12th		
1.	0	0	0	0	0	0	0	0	*
2.	0	*	0	0	0	0	0	*	*
3.	0	0	0	0	0	0	0	*	*
4.	0	0	0	0	0	0	0	0	0
5.	0	†	0	†	†	†	†	*	†
6.	0	0	0	0	0	0	0	0	*
7.	0	*	0	0	0	0	0	*	*
8.	0	0	0	0	0	0	0	*	*
9.	0	0	0	0	0	0	0	*	*

*First period. †Terminal.

We must distinguish between pain and tenderness: the one is subjective and the other objective. The subsidence of pain in a case of subperiosteal abscess, wherein the nerve filaments in the periosteum are bathed in pus and consequently show an inflammatory infiltration, is identical with the subsidence of eye pain when the extradural abscess over the petrous tip produces an infiltration of the Gasserian ganglion. Tenderness, in a case of subperiosteal abscess, is elicited by pressure over the area. This pressure results in a temporary re-establishment of the conditions which produced pain prior to the rupture of the pus through the cortex, namely, an increase in the tension exerted on the nerve fibers in the periosteum. The eye pain will reappear after the petrous tip suppuration has resulted in an extradural abscess, when an increased tension is again exerted on the ophthalmic nerve: this occurs with the invasion of the subarachnoid space. A purulent meningitis results with a marked increase in intracranial tension which is transmitted not only to the ophthalmic nerve on the side of the lesion but to all the sensory nerves of the dura. A severe generalized headache then sets in.

If the patient's life is to be saved and a meningitis averted, operative measures must be instituted prior to the stage of quiescence. In other words, the pus in the tip must be evacuated before it has ruptured into the subdural space. Therefore, the stage of quiescence is not to be utilized as a diagnostic aid in determining

the presence of a petrosal tip suppuration. This will again be discussed when the symptomatology is correlated.

The Terminal Period.—The terminal period presents, in the main, the clinical picture of an acute purulent leptomeningitis. One notes a gradual onset of cervical rigidity, the appearance of photophobia, high temperature, Kernig and Brudzinski, severe generalized headache, projectile vomiting and a purulent spinal fluid.

Whether or not any of the cranial nerves in the proximity of the petrosa will be involved depends upon the point of rupture in the petrous apex. In one of our cases (Case 5) the rupture occurred in the vicinity of the posterior fossa and consequently the terminal stage saw the seventh, ninth, tenth, eleventh and twelfth nerves involved. This patient showed a facial paralysis, a loss of taste and sensation in the pharynx on the side of the lesion, hoarseness due to unilateral fixation of the cord, unilateral paralysis of the soft palate, deviation of the tongue and drooping of the shoulder (see Table 5).

In none of our cases was the abducens nerve involved. This seems incongruous at first, because of the deeply rooted impression that a lesion of the petrous apex produces the so-called Gradenigo syndrome. In the introduction to this paper we have outlined the reasons why the Gradenigo syndrome as such is not diagnostic of a petrous tip suppuration, and we have shown conversely that not all cases of petrous tip abscess produce a paralysis of the sixth nerve.

Uffenorde²⁶ found an abducens palsy in only two of his seven cases. Lange's⁹ two cases had no abducens palsy. Guttich⁴⁷ cites a case of sequestration of the entire pyramid without abducens paralysis. Eagleton's¹³ two cases of caries of the petrous apex showed no external rectus involvement. On the other hand, many cases are on record wherein an abducens paralysis has been found associated with a suppurative lesion in the petrous tip. Because of the fact that abducens palsy is not a constant symptom, in the sense that the retro-orbital pain, low grade sepsis and persistent otorrhea are constant, it is not to be looked upon as a necessary factor in the establishment of a diagnosis of petrous tip purulency. For one thing, the position of the abducens below the

tentorium renders it more easily involved by lesions located in the posterior cranial fossa. In addition, Eagleton's dissections, which have been alluded to in the first section of this paper, show that a number of factors influence the susceptibility of this nerve to lesions which might disturb its function in the vicinity of the petrous tip. The difference in the length of the nerve in Dorello's canal, the extent to which the petrosphenoid articulation is closed, variations in the size of the nerve itself and its freedom in the canal, all help to determine whether or not the nerve will be involved by a suppurative lesion in the petrosal tip.

When we consider that most cases presenting the so-called Gradenigo syndrome go on to complete recovery, whereas cases of petrosal tip suppuration either terminate fatally or result in a chronic otorrhea if no attempt is made to eradicate the lesion surgically, it is inconceivable that the two conditions should be considered identical. Since we have seen that suppurations of the petrosal tip occur without an involvement of the abducens, it is illogical to look for the outward manifestation of external rectus palsy as a symptom diagnostic of petrosal tip suppuration.

It has been our experience that the abducens palsy in the course of an otitic suppuration is most often associated with a mild type of meningeal inflammation. In this opinion we are in accord with Körner,⁵ Alt,⁶ Sears⁸ and others. In other words, while a paralysis of the external rectus may occur in association with a purulent focus in the pyramidal apex, its presence in such cases is the exception rather than the rule. In fact, the presence of abducens palsy is more apt to point against than to the diagnosis of petrosal tip suppuration. We have taken up this point in detail because of the insistence of some of the men who saw some of our cases in consultation with us upon classing them under the category of Gradenigo syndrome. They expressed themselves in favor of waiting for further signs to make their appearance before making a diagnosis of petrous tip suppuration and called attention to the fact that no abducens palsy was present to corroborate our diagnosis. In each instance the eye pain, the low grade sepsis, the persistent otorrhea and extensive pneumatization and destruction of the petrosal tip, as evidenced by radiographic examination, were present. Nevertheless, the absence of external rectus paraly-

sis was considered a significant factor pointing against the diagnosis of petrous tip suppuration. All of these cases were subsequently proven to be petrosal suppurations at the operating table; and the delay caused by waiting for the abducens palsy, which never appeared, resulted in a fatal meningitis which might have been avoided had operative measures been instituted earlier.

Laboratory Data.—The information which the laboratory furnishes in these cases is negligible. Only the X-ray can be used to decided advantage.

Bacteriologic Flora.—There are many reports in the literature which would tend to associate petrosal tip suppurations with the pneumococcus III. Grünert's³⁴ three cases were due to a pneumococcus infection. Leutert's³⁶ case also showed a pneumococcus. Uffenorde²⁶ considers the finding of a streptococcus mucosus in association with extensive pneumatization as a significant factor in the diagnosis of this lesion. The third case of Friesner and Druss²⁸ showed a pneumococcus type III, while the first case yielded a streptococcus hemolyticus.

In our nine cases, six of which we were able to study from the outset of the lesion, and in two of which we had access to the earlier records, none showed a mucosus infection. Two showed streptococcus viridans, one a streptococcus nonhemolyticus. Two had a streptococcus hemolyticus which was not subcultured and the remaining three a streptococcus pyogenes.

It is our opinion that the invading organism has no etiologic relationship to the lesion in question. A streptococcus mucosus infection occurring in a patient who has no pneumatic structure in his petrosa cannot possibly produce a petrosal tip suppuration. This holds true for the other bacterial agents. In other words, the lesion in the petrosal tip depends upon the type of anatomic structure rather than upon the type of bacterial invader.

The tendency to consider that suppuration of the petrosal tip is due to a pneumococcus type III, springs from the fact that in both cases the disease runs a protracted, almost symptomless course until fatal intracranial lesion supervenes. However, if the cases of a petrosal tip suppuration are carefully studied, it is at once seen that definitely diagnostic symptoms make their appearance as soon as the tip is invaded by the suppurative focus.

In the case of an otitic infection caused by the pneumococcus III, the insidious advance of the lesion and the complete lack of prodromal signs are due to the inability of the bodily mechanism to set up successful inflammatory barriers against this organism.⁴⁸

Blood.—The red cells and hemoglobin are not affected to any extent by this lesion. All our cases showed a mild secondary anemia such as is usually associated with any prolonged sepsis. The white cell count, which would be expected, under the circumstances, present in these cases, to manifest a severe infection by a shifting to the left in the v. Schilling count, showed but a slight increase in staff forms, 6 to 9, and a moderate leucocytosis, 10,000 to 13,000. Only toward the terminal stage, when the endocranium was already invaded and a purulent meningitis established, was there a sudden rise in the staff cells, 15 to 19, and a marked leucocytosis, 16,000 to 22,000.

For practical purposes, therefore, the examination of the blood yields no evidence beyond the fact that some infection is still present which is causing an increased leucocytosis. It gives no data concerning the severity of the infection until the terminal stage.

Spinal Fluid.—In all of our cases the spinal fluid showed normal chemical, bacteriologic and cytologic findings prior to the terminal stage, when a purulent meningitis appeared. In the cases which recovered, the spinal fluid remained normal. In only one instance was there any increase in pressure noted by the manometer.

Eyegrounds.—With one exception (Case 3), the eyegrounds in all our cases showed normal discs. In this case a sinus thrombosis had been present, necessitating a ligation of the jugular and an obliteration of the sinus. This might have accounted for the slight blurring of the discs which was noted.

X-Ray.—With the exception of the eye pain, persistent otorrhea and low grade sepsis, this furnishes us with the most valuable guide that we have toward the diagnosis of the lesion. We have been using the regular position for the base of the skull, which gives an undistorted picture of the entire length of the petrous pyramid. In addition to the regular plates for examination of the mastoid process, a base plate is taken in all cases so

that we have a basis of comparison, should we require it. Other observers have reported on the use of the X-ray in diagnosing destruction of the petrous apex. Mangabeira-Albernaz²¹ states that radiographic plates, taken after the technic of Hirtz and Magnien, will show disease of the tip cells. Bigler⁴⁰ utilizes the technic of Stenvers and states that this method shows the disease in the tip cells.

We have attempted, with Dr. Henry K. Taylor, to employ these and other positions in our cases; but, from a diagnostic standpoint, the base plate has furnished us with the best information in every case. It has one objection, that it gives a view of the breadth and length only. Dr. Taylor is at present working to perfect a new position which will enable us to obtain more detail.

In cases which, before the onset of the eye pain, showed an extensive pneumatization in the mastoid process, the base plate likewise demonstrated air spaces within the petrous bone and tip. By comparing the two sides, after the lesion had developed in the tip, a distinct difference could be noted. This varied from a blurring of the cells to complete destruction of the tip. In the section devoted to the case histories these plates will be reproduced.

THE COURSE OF SUPPURATIONS IN THE PETROUS TIP.

A patient has an acute nasopharyngeal infection, due to any one of a number of causes, and then develops an abscess in the ear. Paracentesis is performed; and, after an interval of time, an acute coalescent mastoiditis develops. Radiographic examination reveals an extensively pneumatized mastoid process which has undergone destruction. At operation, a large mastoid cavity is uncovered with diseased cells located not only in the mastoid process but in the zygoma, squamosa, peribulbar area and the region surrounding the semicircular canals.

After a period of normal convalescence, during which the temperature returns to normal, the middle ear ceases to discharge and the mastoid wound heals normally, the patient begins to complain of pain in the eye on the side of the lesion. About the same time there occurs a sudden profuse discharge from the

middle ear, but none from the mastoid wound, which by this time may have entirely healed. The patient begins to run a low grade temperature, 99 in the morning and 101 to 102 in the afternoon. The pain in the eye is complained of mostly at night, and at times the patient has a sensation of dizziness, sometimes occasional vomiting spells. A transient facial weakness may be present.

Examination at this time reveals a profuse otorrhea, which is coming through a central perforation in the drum. There is a slight nystagmus which has no fixed direction. The fundi are normal. The blood count shows only a moderate leucocytosis with but a slight increase in staff cells. An X-ray picture of the petrous pyramid taken now reveals distinct pathologic changes in the region of the pyramidal apex. If the patient is operated upon at this time according to the technic to be described in the next section of this paper, pus will be evacuated from the petrosal tip, which will be found to contain a mass of soft granulations. Operation performed at this stage usually results in recovery.

If the lesion is permitted to advance, the eye pain suddenly subsides and completely disappears for a time. The patient feels well and only the fever remains. After a short time, usually no more than ten days, a fatal meningitis supervenes.

The variations in this picture are not numerous. The discharge from the ear, instead of clearing up and then recurring, may be continuous after the performance of the simple mastoidectomy. In addition to the eye pain, there may be a neuralgia of other branches of the trigeminal nerve. The period of quiescence may be totally absent or of very short duration, so that the eye pain persists until the advent of the terminal meningitis. The case may heal spontaneously by the establishment of adequate drainage from the tip into the middle ear, in which case a chronic otorrhea will result. Whether such cases, of which we have two under observation, will eventually heal completely or whether the necrotic process in the bone will ultimately lead to a fatal intracranial complication, cannot be decided with any degree of certainty at the present time. The fact that one case shows the necrosis to have involved the basilar process of the occipital bone

tends toward the idea that eventually the patient will succumb to a basilar meningitis.

In any event, it is our opinion that all of these cases should be operated upon and adequate drainage established for the pus in the petrous pyramid. If operative measures are instituted early, many of these cases will be saved the fatal meningitis which is the usual outcome of a petrosal tip suppuration.

DIFFERENTIAL DIAGNOSIS.

Suppurations of the petrous tip must be differentiated from nasal accessory sinus disease, lateral sinus thrombosis, cavernous sinus thrombosis, thrombosis of the superior petrosal sinus, acute labyrinthitis and a reinfection of the mastoid process.

Nasal accessory sinus disease, when the frontal and sphenoid sinuses are involved, may very often simulate a petrous tip suppuration in the presence of an aural discharge. Both will give pain in the region of the eye. A sphenoiditis, particularly, may, by a neighboring edema of the cavernous sinus, give deep seated eye pain. In the absence of aural discharge, involvement of the petrous tip can be immediately excluded. Where an aural discharge is present, however, probing the sphenoid sinus, posterior rhinoscopic examination and X-ray of the sphenoid will exclude this lesion. The same means are employed for ruling out a frontal sinusitis.

A cavernous sinus thrombosis is not a common complication of an aural suppuration. Where it occurs, it is the result of an extension of a thrombosis in the sigmoid sinus into the cavernous. It is characterized by a septic temperature, positive blood culture, chemosis of the conjunctiva, Crowe-Beck sign, third, fourth and sixth nerve paralysis. In the presence of these findings, pain behind the eye can be ruled out as designating a petrosal tip suppuration.

Thrombosis of the superior petrosal sinus also results from the extension of a thrombus located in the sigmoid sinus. In its extension this also may give rise to an inflammatory reaction in the neighborhood of the Gasserian ganglion and sixth nerve. Its presence should be suspected in a case with eye pain when sepsis

continues after removal of the thrombus in the lateral sinus and blood culture is positive. In this lesion the discharge from the middle ear has not the characteristics described for suppurations of the petrous tip. Occasionally an atypical clinical picture of lateral sinus thrombosis gives fifth nerve pain with absence of the classical picture of sinus sepsis. Here the assembled data from the roentgenogram of the tip helps the differentiation and locates the lesion in the pyramid.

In the presence of vertigo and nystagmus, an acute labyrinthitis must be ruled out. This is easily done by the functional tests of the labyrinth. These tests yield normal findings in a case of petrosal suppuration, whereas, in acute labyrinthitis, findings varying from a hyperirritability to a complete loss of response will be found. Furthermore, the patient suffering from acute labyrinthitis assumes the forced position noted in such cases.

A petrosal pyramid suppuration is differentiated by the following factors from a reinfection of the mastoid wound, which a renewed discharge from the middle ear immediately suggests. It shows no clinical manifestations of a renewed inflammatory process in the mastoid wound. There is no swelling of the incision, no increase in wound discharge. After the canal has been wiped dry, pressure over the recently healed scar does not cause pus to pour into the middle ear, showing that the source of the pus must be at a point remote from the mastoid wound. There is no marked increase in temperature, such as usually accompanies a reinfection. There is no pain referable to the wound and no undue tenderness beyond what one ordinarily elicits on pressure over a fresh scar.*

(TO BE CONTINUED.)

51 WEST 73RD STREET.

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XIX.

CEREBELLAR PONTILE ANGLE TUMOR, WITH REPORT OF A CASE.*

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OMAHA.

Tumors of the cerebellopontile angle are of especial interest to the otologist for three reasons. First, because the early symptomatology frequently is suggestive of an inner ear lesion. Second, because the accurate diagnosis or suspicion of such a condition may lead to surgical attack and cure. And third, because of the assistance the otologist may give as a consultant in making a differential diagnosis in those cases which do not come first under his observation.

As regards incidence, tumors of the cerebellopontile angle may be considered as rare. And yet Cushing's statistics indicate that 7 per cent of all intracranial growths originate in this locality. Numerous isolated reports and a few systematic studies of the lesion have been made since the first accurately recorded case described by Charles Bell in 1830. But the comprehensive monograph by H. Cushing in 1917 concerning tumors of the nervus acusticus was the first analysis of the subject to the exclusion of other lesions in the cerebellopontile recess, and it is to this work that we naturally turn for reference as to the nature of the growth and the symptom complex which it induces.

These growths generally arise from the distal portion of the eighth nerve as it is about to enter the internal auditory meatus, and as they grow, push into the anterior fossa producing pressure upon adjacent structures. Histopathologic study classifies the tumors as of true nerve tissue origin—that is, either neurofibroma or neuroglioma. They are nonmalignant and are definitely encapsulated by the arachnoid. Either sex is equally susceptible, but age is an etiologic factor in that most cases occur after the

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age of twenty, the majority being discovered in persons between the ages of fifty and sixty. A few rare cases of bilateral growths have been reported. The average duration of unoperated cases from the time of onset until death is from four to six years.

The symptomatology may be grouped into three divisions. First, those referring to function of the eighth nerve itself; second, those suggestive of cerebellar disturbances, and third, the effects of pressure upon adjacent cranial nerves.

As would be expected from the anatomic location, the first symptoms to attract attention are those arising from disturbance of the nervus acusticus on the same side of the tumor. Very early in the process, tinnitus presents as an irritative manifestation followed by deafness as a paralytic manifestation of involvement of the cochlear portion, and with an implication of the vestibular portion, vertigo and ataxia. Since there are but few exceptions to this mode of onset, a careful history is of paramount importance in differential diagnosis, for the reason that, if symptoms of affection of the eighth nerve make their appearance some time after other neurologic disturbances, the lesion is in all probability not of acoustic nerve origin.

Since the symptoms of tinnitus and progressive deafness sometimes antedate all others by several years, the possibility of such a tumor should be considered when a patient presents such symptoms, and he should be given the benefit of a careful neurotologic examination, particularly when the symptoms are on one side only.

The disturbance of function of the vestibular portion is manifested by vertigo, instability and nystagmus. The vertigo may occur in attacks and usually is described as a rotation of self or of surrounding objects. The nystagmus generally is horizontal and aggravated when the patient looks toward the side of the lesion. As progression occurs, vertical nystagmus frequently appears, due to pressure on the brain stem.

Cerebellar symptoms are first due to pressure, later to actual invasion of cerebellar substance. And unless the case comes under observation early, or if the chronology of events is not carefully considered, the picture may be more that of an actual cerebellar lesion than of one originating in the angle. Briefly, cerebellar

symptoms constitute, (a) unsteadiness of the lower extremity on the side of the lesion to a greater extent than that of the corresponding arm, (b) there is a tendency to fall to the side of the lesion or to deviate to that side when walking, (c) tremor of the upper arm and a tendency to drop objects from the hand. Variations in the intensity of the above symptoms may occur from time to time in the same case.

Involvement of the other cranial nerves is generally considered to be a relatively late manifestation. The fifth, sixth and seventh nerves are the ones usually affected. Slight numbness or a tingling of the face or corneal anesthesia refer to the fifth nerve; interference with the motor division of the fifth may produce weakness of the masticatory muscles, also deviation of the jaw to the affected side. Double vision or demonstrable weakness of the external rectus follows sixth nerve pressure; lagging of the facial muscles or a slight flattening of the lines of the face without loss of voluntary control results, of course, from involvement of the seventh nerve.

Terminal symptoms include those which we naturally associate with increased intracranial pressure—choked disc, intense headache, vomiting and perhaps slow pulse. Paroxysmal pain in either the occipital or frontal regions and tilting of the head toward the affected side may, however, occur early in the course of the process.

The part which the otologist plays in the diagnosis of these rare tumors naturally depends upon his analysis of the function or dysfunction of the eighth nerve and its end organs. The progress of the last twenty years has given us means of investigation which permit more accurate diagnoses than the symptomatic ones of Ménière's disease, Jacksonian epilepsy, progressive bulbar paralysis, etc., which were common prior to this period.

Naturally, careful study of the cochlear portion of the nerve is important, but it is principally upon vestibular reactions to caloric and turning stimulations that we depend for information. In the incipient stage diagnosis is not always easy, but in the early manifest stage the findings are often so characteristic as to be pathognomonic. Here we expect to find a diminution or absence

of response to caloric stimulation from the horizontal and vertical canals on the affected side. On the contralateral side the horizontal canals always respond, but the frequent absence of reaction from the opposite vertical canals is an extremely important and suggestive finding.

The case I wish to report regards a man, age 49, a farmer by occupation, sent to the University Hospital November 17, 1930, with a tentative diagnosis of Meniere's disease. His complaints, which were of one year's duration, consisted of dizziness, headache, nausea and vomiting, and occasionally tinnitus. On arising in the morning vertigo was at once in evidence, and was then followed by nausea and vomiting which relieved the headache, but not the dizziness. The headache was described as a sensation of occipital pressure. About six months prior to admission he noticed a tendency to deviate to the left when walking and a tendency to fall to the left when standing with eyes closed. At approximately the same time he remarked that his knees tended to go out from under him and he had a weakness of the left arm. Almost coincident with the onset of vertigo he noticed a marked reduction of the hearing in the left ear, which rapidly progressed to extreme deafness.

Previous history elicited nothing essential other than the loss of twenty pounds of weight during the past year.

When physical examination was instituted the individual was lying prone in bed with no active complaints. When asked to sit up he did so, slowly and complaining of vertigo. When he stood, he held the left shoulder high, the head was somewhat turned to the left. The feet were placed widely apart and he was unable to stand without swaying, occasionally requiring support. Physical examination, except for findings pertaining to the nervous system, was negative.

Neurologic examination revealed the following positive findings: third degree of deafness in the left ear; spontaneous horizontal nystagmus when looking to the left; occasional vertical nystagmus when looking up; positive adiodokokenesis of left arm; ataxia in finger—nose test on the left side; motor weakness of both left upper and lower extremities; ataxia of the left lower extremity. His gait was ataxic with a tendency to deviate to the left, putting his feet down carefully and walking on a wide base.

Vision, with correction, was normal. The visual fields and ocular fundi showed no divergence from the normal.

A roentgenologic study of the skull and mastoid regions of each side showed no gross difference in the size of internal auditory meatuses; nor was there any change in the petrosal ridge noted. Special study relative to vestibular function showed a spontaneous horizontal nystagmus, becoming much coarser when looking to the left; a faint horizontal nystagmus when looking to the right. When the eyes were directed upward, a vertical nystagmus was produced. Spontaneous past pointing to the left by the left arm was a variable finding. A positive Romberg, with falling to the left was present. When the head was turned to the

left he tended to fall backward; when the head was turned to the right he tended to fall forward.

Stimulation of the horizontal canals by turning in the Barany chair, first turning to the right (thus giving the greater stimulus to the left labyrinth) gave a nystagmus time of seventeen seconds. This is a reduction of ten seconds below the normal. The nystagmus was of normal type, but there was a total absence of past pointing. Turning to the left, thus giving the greater stimulus to the right labyrinth, produced normal nystagmus of twenty-five seconds duration and, with it, normal past pointing. Stimulation of the right vertical canals produced normal rotatory nystagmus, but when the left vertical canals was stimulated, a very significant variation was discovered. Instead of a rotatory nystagmus to the left, as would be expected, the response was a horizontal nystagmus to the right. In other words, an inverse and perverted nystagmus suggestive of brain stem pressure was produced.

Examination of the right labyrinth by the caloric method gave normal response from the horizontal canal, but an absence of response from the vertical canals—further evidence of brain stem involvement. Douching of the left ear produced no nystagmus from the horizontal canal and only a slight twitching of the eyes when the head was in position for vertical canal response. Past pointing was absent except for a tendency of the right hand to past point to the left.

After analyzing the foregoing information, a diagnosis of left cerebellopontile angle tumor, producing pressure upon the pons, seemed justified for the following reasons:

1. Chronology of events.
2. The general neurologic findings.
3. Extreme left sided deafness.
4. Spontaneous vertical nystagmus and spontaneous horizontal nystagmus, principally to the left.
5. Marked reduction of all left semicircular canal responses—plus an inverse and perverted nystagmus when the left vertical canals were strongly stimulated.
6. Normal response from the right horizontal canals, but diminished response from the right vertical canals.

The patient was operated on by Dr. J. J. Keegan of Omaha on December 1st, 1930, who found a tumor in the anticipated locality producing pressure upon the pons and cerebellum as evidenced by displacement to the right and herniation of the cerebellum into the foramen magnum.

The patient made a satisfactory postoperative recovery and was dismissed from the hospital December 21st, 1930, in good condition although still moderately ataxic.

1620 MEDICAL ARTS BLDG.

XX.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES: OBSERVATIONS IN EIGHTY-ONE CASES WITH SPECIAL REFERENCE TO OVERLOOKED FOREIGN BODIES.*

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The subject matter of this paper is based upon observations made in 81 foreign body cases from the private practices of my associate, Dr. J. B. Naftzger, and myself. At this time I wish to give some general statistics of the entire group, discuss in some detail overlooked foreign bodies and delayed diagnosis in fifteen cases, and make some remarks on results of treatment in individual cases. The object of the paper is not to present anything new in diagnosis or treatment but rather to emphasize again the importance of foreign bodies in the consideration of obscure chest conditions. The number of overlooked foreign bodies and wrong or delayed diagnoses is, in my opinion, much too high, especially when very often the history alone, with possibly the help of the X-ray, is all that is necessary to clear up the diagnosis of an apparently obscure case.

GENERAL STATISTICS.

Types and numbers of foreign bodies:

FOREIGN BODIES IN AIR PASSAGES		FOREIGN BODIES IN ESOPHAGUS	
Corn kernels	11	Nickel	4
Peanut	6	Penny	4
Sandburr	5	Dollar	2
Lima bean	1	Safety pins	6
Apple core (portion)	1	Straight pin	1
Almond	1	Piece of brass	1
Fishbone	1	Chicken bones	7
Prune pit	1	Iron washer	1
Watermelon seed	3	Prune pit	1
Brazil nut shell	1	Chicken gizzard	1
Hazel nut	1	Cherry stone	1

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Meat	1	Meat	1
Egg shell	2	Thumb tack	1
Celluloid	1	Button	3
Tooth	1	Screw	1
Mucous (possibly small foreign bodies)	3	Pork chop bone.....	1
Nail	1	Squab bone	1
Straight pin	1	Oyster shell	1
	42	Metal wheel from toy.....	1
			39

The foreign bodies were present in the larynx, trachea, bronchi or esophagus.

There were 42 in the air passages and 39 in the esophagus. Corn kernels appeared eleven times and peanuts six in the trachea or bronchi. Sandburrs were removed from the larynx five times with the laryngoscope and in several other instances with the cocain applicator.

In three cases "mucus only" listed because foreign particles were too small to identify. History and symptoms suggestive of foreign body. In the esophagus group chicken bones appeared eight times, coins eight times and safety pins six.

The time of sojourn of the invader in the air passages varied from ten minutes to 105 days; in the esophagus from one hour to eight months.

The foreign body was overlooked for days, weeks or months in fifteen instances. In eight of this number there was definite history of foreign body ingestion, and in the others there was invariably sudden onset with very suggestive foreign body symptoms.

Seventy per cent of the patients in the tracheal and bronchial group and 20 per cent in the esophageal group were three years of age or under.

Where the trachea or bronchi were explored, lower bronchoscopy with tracheotomy was used three times; upper bronchoscopy without tracheotomy thirty times.

Tracheotomy following bronchoscopy was found to be necessary in two patients.

Tracheotomy was necessary before successful esophagoscopy in one instance.

In the 81 cases ether was the anesthetic twenty times, cocain twenty-three times and chloroform once.

No anesthetic was used in 37 procedures, and in this group 32 of the cases were five years or under and none were over ten.

There were five deaths during or after endoscopic procedure in the 81 cases, two of them after bronchoscopy and three after esophagoscopy.

In two patients foreign body removal failed the first time, and lung abscess developed with, however, recovery after subsequent developments.

One stricture of the esophagus followed where a foreign body had been in the esophagus eight months, but a good result was obtained after several dilatations with heated bougies.

The final result in each patient of the entire group was either death or complete recovery.

OVERLOOKED FOREIGN BODIES.

In this series there were fifteen cases in which the possibility of foreign body was not considered, though very definite and suggestive symptoms had been present from a few days to eight months. In the bronchial group of eight such cases, there was definite history of foreign body ingestion in seven, yet no attention was paid to this history for days, weeks or even months. Many and often fanciful diagnoses were made and very serious symptoms developed in most instances before the proper diagnosis was made. Several times patients were brought for bronchoscopic study by the parents or friends against the advice of the attending physician. In the esophageal group only once in a group of seven cases was the history definite of foreign body ingestion, but many of the symptoms were practically diagnostic for weeks and even months. It is our duty to disseminate information whenever possible to the medical profession that foreign bodies in the air and food passages are much more common than is usually believed. With the possibility in mind, the history and symptoms make the probable diagnosis of foreign body very simple in many instances. How many patients go to their death, because of unrecognized foreign bodies in the bronchi especially, can only be surmised. Certainly it seems logical to suspect that a certain small percentage of all bronchopneumonias in children have a foreign body origin.

AIR PASSAGES.

Type of Foreign Body	Case No.	Interval between ingestion and removal	Diagnosis	Previous history of foreign body ingestion
1. Eggshell	80	6 weeks	a. Bronchitis b. Asthma c. Common cold	Definite (eggshell in mouth and feces)
2. Peanut	69	9 weeks	Pneumonia—3 separate attacks	Definite (eating cracker-jack)
3. Corn kernel	33	3 days	None made	Definite (corn kernels in mouth)
4. Eggshell	54	3 days	Diphtheria (laryngeal)	Definite (crushed eggshell in hand)
5. Peanut	31	9 weeks	a. Pneumonia b. Empyema (rib resection) c. Tuberculosis	Definite (eating salted peanuts)
6. Prune pit	10	3½ months	None made (pronounced negative by 3 physicians)	Definite (eating prunes)
7. Corn kernel	16	12 days	Common cold	Definite (playing with shelled corn)
8. Peanut	11	2 days	Pneumonia	Not definite but sudden onset while playing

ESOPHAGUS.

Type of Foreign Body	Case No.	Interval between ingestion and removal	Diagnosis	Previous history of foreign body ingestion
1. Cherry	58	6 weeks	Lye stricture	Not definite but sudden onset after years of difficulty from lye stricture.
2. Piece of brass	20	8 months	a. Stomach trouble b. Tonsils and adenoids c. Bronchitis d. Asthma	Not definite but sudden inability to swallow solid food.
3. Vertebra of chicken	51	2 weeks	Negative (after stomach tube and esophagoscopy)	Definite (eating chicken sandwich)
4. Button	24	2 months	Sore throat and cold	Not definite but choked suddenly and no solid food afterwards.
5. Button	60	2 weeks	a. Throat infection * b. Enlarged thymus	Not definite but onset sudden with choking spell.
6. Screw	18	3 days	Diphtheria (laryngeal)	Not definite but onset sudden with inability to swallow, plus croupy cry.
7. Dollar	74	30 days	No previous examination	Not definite (unable to swallow solid food after intoxication);

A short abstract of these overlooked foreign body case reports will probably tell this story better than mere statements:

AIR PASSAGES.

Case No. 31.—History: Child, age five, had severe choking spell, nine weeks previous to our examination, while eating salted peanuts. Difficult breathing followed. Temperature 104 by next day, dyspnea and cough. Physician diagnosed pneumonia left lung. Ten days, patient worse, diagnosis empyema and rib resected but no pus found. Septic temperature, loss of weight, strength. Emaciation. After five weeks in bed diagnosis of tuberculosis was made. During entire course foreign body was not considered except by parents. X-rays of chest were thought to rule out foreign body.

Examination and treatment: Emaciated patient, no expansion left chest, feeble breath sounds over left apex, absent over rest of lung. Right lung emphysematous.

X-ray of chest: Drowned lung left.

Bronchoscopy without anesthesia: Many pieces of peanut found at opening of left bronchus, muco pus released, removed by suction. Secondary bronchoscopy in four days. Rapid and complete recovery.

Case No. 69.—History: Child, age 22 months. Nine weeks before our examination choked while eating cracker-jack. Cough, difficult breathing, peculiar rattle in chest described by parents. Next day temperature 104 with cough. Physician diagnosed pneumonia. Septic course for nine days with wheezing respirations, then improvement. During next seven weeks had two more severe attacks with fever, cough and dyspnea with diagnosis of pneumonia each time.

Examination and treatment: Patient was brought for bronchoscopic study by the parents without the advice of the family physician. Upper bronchoscopy without anesthetic. Large single piece of peanut found at opening of left bronchus, apparently had been free in trachea much of the time. Foreign body removed. Rapid and complete recovery in short time.

Case No. 54.—History: Child, age 6 months. Three days before our examination baby had a severe choking spell. Crushed eggshell was found in its hand. Hoarse cry, difficult breathing developed immediately. Family physician diagnosed diphtheria and two doses of antitoxin were given. Difficult breathing became worse, was sent in for possible tracheotomy and treatment as diphtheria.

Examination and treatment: Baby in great distress with difficult breathing of laryngeal type. No cry.

Laryngoscopy without anesthetic: Several pieces of eggshell removed from larynx below and between cords. Recovery rapid without tracheotomy.

Case No. 80.—History: Child, age one year. Six weeks previous to our examination baby choked. Pieces of eggshell were present in the mouth (some pieces later were passed by the bowel). Patient developed hoarse cry, constant difficult breathing, cough. Three physicians treated

baby for asthma, bronchitis and common cold. Fourth physician took X-ray and reported it negative for foreign body. Otolaryngologist heard history, advised examination of larynx.

Examination and treatment: Some cyanosis, laryngeal stridor, hoarse cry, breath sounds clear each lung.

Laryngoscopy without anesthetic: True and false cords badly swollen. Pieces of eggshell removed from just below cords. Tracheotomy was necessary next day. Complete recovery.

Case No. 10.—History: Child, age 7 years. Three and one-half months before our examination child had a severe choking spell while eating prunes. Breathing and swallowing difficult for two days. Severe choking spells with cyanosis at intervals with only mild wheezing respiration between these attacks. General condition good. Several physicians examined the child, reported the chest negative, paid no attention to the history. Finally otolaryngologist sent patient for bronchoscopic examination after 3½ months of symptoms.

Examination and treatment: Breath sounds clear over lungs, but definite wheezing over trachea. Fluoroscopy and X-ray of chest negative. Audible tracheal slap when patient coughed was definite.

Laryngoscopy and bronchoscopy with ether: Prune pit found wedged in opening of right bronchus and removed. Much mucus removed from trachea by suction. Rapid and complete recovery.

Case No. 33.—History: Child, age 3 years. Three days previous to our examination mother found child in a severe choking spell. There were several kernels of corn in his mouth. Wheezing respiration with cough and cyanosis at times. Parents took the child to three different physicians and each one after examination of the patient insisted there was nothing wrong. No attention was given to the definite history.

Examination and treatment: Hoarse cry, wheezing respiration, croupy cough. Poor expansion left chest. Feeble breath sound entire left chest. Right lung emphysematous. X-ray: Faint blurring entire left lung.

Upper bronchoscopy without anesthesia: Kernel of corn wedged tightly in opening left bronchus removed. Recovery uneventful.

Case No. 11.—History: Child, age 22 months. Thirty-six hours before our examination child was struck in the face by an auto tire used as a swing. Child coughed, cried and became cyanotic. Condition improved some for twelve hours, then had severe choking spell and difficult breathing. Cyanosis marked. X-ray of chest taken which showed complete blur of left chest.

Examination and treatment: Patient was brought to the bronchoscopic table in extremis. Very marked cyanosis. Very shallow respiration.

Upper bronchoscopy and artificial respiration: Large amount of secretion came from the bronchoscope. Small pieces of peanut found in this discharge. Suction and artificial respiration failed to revive child. Much swelling opening left bronchus.

Comment: X-ray and findings indicated very marked case of drowned lung. Foreign body probably responsible for condition. This is the only case in this group which did not have a definite history suggesting foreign body ingestion.

Summary: In this group of eight overlooked foreign bodies in the air passages, there was a definite foreign body history in seven. Foreign bodies were present from a few days to $3\frac{1}{2}$ months. In two cases peanuts were present for nine weeks. In all cases symptoms were so strongly suggestive that foreign body should have been considered early.

FOOD PASSAGES.

Case No. 20.—History: Child, age 3 years. Eight months before our examination began choking on solid food. Could swallow liquids well. No trouble with respiration. Kept on liquid diet for four months and treated by a physician for stomach trouble. Loss of weight and strength. Six months after first symptoms tonsils and adenoids were removed by an otolaryngologist to relieve the difficult swallowing. During next month some difficult breathing and cough in addition to difficult swallowing. Was treated by another physician for bronchitis and asthma. Sore throat, dyspnea, fetid breath developed. Much emaciation. Finally X-ray of chest by fourth physician. Large metallic foreign body in esophagus.

Examination and treatment: Upper esophagoscopy without anesthesia. Piece of brass from the timer of Ford car removed from ulcerated upper esophagus. Stricture developed. Several dilatations with heated bougies relieved stricture.

Case No. 24.—History: Child, age 3 years. Two months before our examination had a severe choking spell. Unable to swallow solid food since. Kept on liquid diet but no examination was suggested to find cause of difficult swallowing. Difficult respiration, cough and laryngeal stridor prompted bringing child for examination.

Examination and treatment: Emaciated patient. Marked dyspnea, rapid pulse. Lungs clear.

X-rays: Foreign body, apparently button upper third esophagus.

Upper esophagoscopy: Ether anesthesia. Button removed from ulcerated upper esophagus. Much necrotic tissue. Larynx and trachea pressed forward.

Comment: Ether is dangerous in such a case and should not have been used. Complete recovery without stricture.

Case No. 18.—History: Child, age 3 months. Baby became suddenly croupy three days before our examination. Cough, difficult breathing and swallowing. Physician diagnosed diphtheria and gave antitoxin. Sent to the hospital for possible tracheotomy.

Examination and treatment: X-rays. Large screw upper part of chest.

Laryngoscopy: No anesthesia. Very large ordinary screw found in upper esophagus and removed. Foreign body was causing great pressure on larynx. Rapid recovery.

Case No. 51.—History: Age 32 years. Two weeks before our examination choked while eating a chicken sandwich. Difficult swallowing developed and persisted. Three days after first choking spell patient was examined by an otolaryngologist who passed an esophagoscope and a stomach tube and reported absence of foreign body. Patient developed

pain in right shoulder and a swollen stiff neck in addition to difficult swallowing. Liquids and some semi-solids could be swallowed fairly well.

Examination and treatment: X-ray of chest. No evidence of foreign body. Tenderness and some swelling anterior and posterior cervical regions. Neck quite stiff. Temperature 100. Fetid breath.

Upper esophagoscopy: Cocain anesthesia. Ulceration and fetid discharge below cricoid. Foreign body removed with difficulty. Proved to be a whole vertebra of a chicken. Recovery rapid without complications.

Case No. 58.—History: Child, age 6 years. Six weeks previous to our examination developed marked difficulty in swallowing. For 2½ years there had been some difficulty in swallowing solid foods because of a lye stricture. For six days before coming under our care child had been unable to swallow water. Had been having rectal feedings. Trouble with swallowing was supposed to be due to the old lye stricture. There was no history of foreign body ingestion.

Examination and treatment: Patient extremely emaciated and weak. Rectal feedings.

Fluoroscopy with thin barium mixture: Complete obstruction of esophagus in middle third.

Esophagoscopy: No anesthesia. Stricture encountered at junction of upper and middle thirds of esophagus. No foreign body evident. Small catheter passed through stricture and water introduced, but would not pass. Procedure discontinued after few minutes because of poor condition of patient. Child to bed with rectal feedings under care of pediatrician. Death same night.

Autopsy: Strictured area three inches long beginning middle one-third of esophagus. Cherry pit solidly impacted two inches from upper stricture bands.

Comment: Child had literally starved to death because of a foreign body which had become impacted in an old lye stricture. This stricture was considered responsible for the sudden and complete obstruction of the esophagus. Esophagoscopy was not advised until child was practically moribund.

Case No. 60.—History: Child, age 15 months. Two weeks before our examination child had a choking spell. Unable to eat solid food after this initial trouble. Several days before coming to hospital developed croupy cough and fever.

Examination and treatment: Chest negative. Marked dyspnea. Fluoroscopy and X-ray negative for foreign body but positive for enlarged thymus.

Laryngoscopy without anesthesia—acute laryngitis.

Bronchoscopy without anesthesia—trachea and bronchi negative.

Upper esophagoscopy attempted. Some edema and inflammation of upper portion esophagus. Procedure stopped because of labored respiration. Second attempt to examine esophagus failed. Patient put in croup tent, breathing became more labored, cough, fever. X-rays: second time reported two plus thymus.

Tracheotomy seven days after first examination.

Upper esophagoscopy: Ulceration and fetid odor. Button found buried in posterior portion of upper esophagus. After tracheotomy could force larynx forward and really examine esophagus, which made procedure comparatively simple. Recovery complete and uneventful.

Comment: Tracheotomy and examination of esophagus probably should have been done much earlier with such definite foreign body history and symptoms. Foreign body was really overlooked for seven days under our care. X-ray of enlarged thymus given too much importance.

Case No. 74.—History: Age 30 years. Patient came to internist to have his chest examined because of difficulty in swallowing solid food, some cough and pain in chest. Four weeks previous to examination developed difficult swallowing after having been intoxicated for twelve hours. Patient thought he had burned his throat with poor liquor.

Examination and treatment: X-ray of chest: Large metallic foreign body in upper third esophagus.

Upper esophagoscopy: Cocain. Dollar removed from upper third of esophagus.

Summary: In this group of seven esophageal cases there was definite foreign body history in only one, but sudden onset in all with symptoms strongly suggesting foreign body. Foreign bodies were present from a few days to eight months before proper diagnosis was made, and often after several wrong diagnoses and much unnecessary waiting.

ANALYSIS OF RESULTS.

	Cases	Recovery	Death	Complications
Bronchoscopy	33	31	2	Lung Abscess (2)
Esophagoscopy	39	36	3	Stricture (1)
Laryngoscopy	9	9	0	
	81	76	5	3

FATALITIES.

1. Case No. 1.—Tracheotomy and lower bronchoscopy on child 2½ years. Small amount of ether used. Portions of lima bean removed from left bronchus. Operation in country. Death six hours later. Proper after care in hospital probably would have saved life.

2. Case No. 11.—Upper bronchoscopy—no anesthetic—child of 22 months. Brought to operating room in moribund condition. Profound cyanosis. Mucus ran out of bronchoscope. Suction used, peices of peanut obtained. Artificial respiration. Death on table. X-ray taken day previous showed complete drowned lung on left. Diagnosis had been pneumonia. Child practically dead when bronchoscopy was done.

3. Case No. 22.—Esophagoscopy. Ether. Child, 15 months. Coin in esophagus disappeared in a pouch each time scope approached. Operation discontinued because of time limit. Next day another attempt with help of fluoroscopy failed after rather long procedure. Patient died twelve hours later, apparently from shock and exhaustion. Long procedure with two anesthetics undoubtedly caused death, which could have been prevented by observing time limit.

4. Case No. 58.—Esophagoscopy. No anesthetic. Child, 6 years. Came to hospital badly emaciated and dehydrated. Difficult swallowing for six weeks. Practically nothing for six days. Fluids by rectum. All trouble had been blamed on old lye stricture. Stricture found upper portion middle third. No foreign body evident. Procedure stopped because patient in very poor condition. Died that night. Autopsy disclosed cherry tightly wedged in old lye stricture. Child died of starvation and dehydration. Esophagoscopy required only a few minutes without anesthetic.

5. Case No. 70.—Esophagoscopy. No anesthetic. Child, 4 years. Very large open safety pin, point up in middle esophagus. Temperature 102 and leucocytosis 23,700 on arrival. Because of size of pin, small child and evidence of sepsis, no attempt made to close pin, but to carry it to stomach. Child breathing very poorly and efforts discontinued. Mediastinitis developed. Right chest became filled, pushed heart to left. Rib resected and pleura drained. Finally died after three weeks of observation. Only one short effort made to reach safety pin.

COMPLICATIONS.

1. Case No. 50.—Lung abscess developed after incomplete removal of kernel of corn by upper bronchoscopy without anesthesia. Patient disappeared from observation after first bronchoscopy when a number of pieces of corn kernel were removed. Returned with a definite lung suppuration. Second bronchoscopy with removal of pus by suction. The firm outer shell of a corn kernel appeared in the secretions and was removed. Gradual but complete recovery.

2. Case No. 49.—Lung abscess developed after unsuccessful attempt to remove a tooth from left bronchus by upper bronchoscopy without anesthesia. Boy of ten years, poor mentality, very obstreperous, unable to control properly without anesthesia. Parents would not allow another attempt with anesthetic and took patient home. Evidence of lung suppuration developed, but parents decided to try Christian Science. After three months of lung abscess symptoms, tooth was expelled during fit of coughing. Complete recovery.

SUMMARY.

General observations based upon experiences with this group of cases:

1. Careful diagnosis of type and location of foreign body of utmost importance as first measure in treatment. Emergency work is seldom necessary, and, unless definitely indicated, hurry is extremely dangerous. Many foreign bodies will change position in a short time, especially when located in the esophagus. Previous X-rays should not be trusted for location of foreign body, but where metallic object is present X-ray or fluoroscopy should immediately precede endoscopic procedure. At times it may be very difficult to decide if foreign body is in the trachea

or the esophagus. Several X-ray plates and lipiodol are often helpful.

2. Proper instruments and these instruments in working order cannot be too strongly emphasized. Experimental work with duplicate of foreign body may save valuable time and help decide proper procedure. Time is of too much importance in foreign body work to allow carelessness to exist.

3. Teamwork wherever possible is of utmost importance. Two surgeons with at least two trained nursing assistants are necessary for the proper handling of many of these cases. The operator using the scope should not have the responsibility of controlling the patient or having proper forceps available at the right instant. Relief of tired eyes and arms by a coworker may change the entire outcome in some instances.

4. Whether or not to use anesthesia is an individual problem in each case. Our policy is to use no anesthesia in small children with a foreign body in the air passages. In esophageal cases in infants our policy is the same, but in older, and especially in obstreperous children, ether is at times chosen with no regrets. Probably there is nothing of more importance in foreign body work than proper judgment in the choice of whether or not anesthesia should be used and for how long.

5. Tracheotomy preceding bronchoscopy was done in this series three times and only in the earlier cases. Thirty consecutive upper bronchoscopies without preliminary tracheotomy have been done in recent years. Two tracheotomies following upper bronchoscopy have been done and both of them came during this past year within a few weeks of each other. Previous to these instances none had been found necessary for ten years. Possibly in impacted laryngeal foreign bodies in infants, with marked reaction in the tissues and in round loose objects in the trachea, preliminary tracheotomy should be considered good judgment. In the ordinary cases we feel that this procedure is unnecessary and adds greatly to the danger in many instances, especially in infants. Procrastination in deciding on tracheotomy after bronchoscopy if patient's condition indicates it cannot be too severely condemned.

6. Time limit on procedures with proper relation to age and condition of patient plays a very prominent part in results. One of our fatalities was undoubtedly due to two prolonged esophagoscopies under anesthesia in a case wherein the scope continually persisted in overriding a coin. On the other hand, some of our good results unquestionably followed because the time of procedure was limited and secondary bronchoscopy was done later. Secondary bronchoscopy is often of great value anyway in allowing removal of thick secretions to relieve embarrassed respiration and allow search for possible multiple foreign bodies.

7. "Mucus only" is listed as a foreign body in three cases. In these patients there was a suggestive foreign body history or symptoms, but no foreign bodies were evident in the secretions except small indefinite particles. However, rapid recovery ensued following removal of this material. Such cases suggest to us that more bronchoscopies should be done in many so-called bronchopneumonias in infants, regardless of foreign body history.

8. The type of foreign body encountered and the relation to the reaction of the tissues have been of interest. We have found the reaction in the mucosa of the trachea and bronchi more marked where the corn kernels were the foreign bodies than where peanuts were, contrary to the usual findings of most observers. There were only six peanut cases, but in none was there any appreciable edema of the mucosa, and two children had carried the foreign body for nine weeks. In the corn kernel cases (eleven) there was usually marked swelling of the vocal cords and often edema so severe as to close the opening of one bronchus. Perhaps these hard objects were loose in the trachea most of the time and traumatized the mucosa during coughing spells. Several times we removed the kernel from the opening of a bronchus, but felt sure it had become impacted there during the introduction of the bronchoscope.

Sandburrs (five) were found in the larynx with the sharp projections in the cords, except in one instance where the offender was in the trachea. Several times this type of foreign body was removed with the cotton applicator during cocaineizing, which, of course, suggests the need for great care in handling such patients

previous to laryngoscopy. Naturally they should be lying down with the head lowered.

Chicken bones (eight) in the esophagus have all been encountered since a report of our cases in 1925. Five were of the thin, flat triangular type so easily overlooked by the esophagoscope, especially as it proceeds toward the stomach. Several of these were only seen as the scope was slowly removed. X-ray was of no help in any of these cases, and the symptoms were very mild, usually only slight difficulty in swallowing with possibly slight pain in the shoulder or back.

Two of the ten coins found were silver dollars. One had been present in the upper esophagus of a young Indian for four weeks. He thought his symptoms were due to burning his throat with poor liquor while he was intoxicated. There was no memory of swallowing the coin.

Safety pins appeared six times and only one caused trouble. In this case the pin was a very large one in a small child. Symptoms were present of sepsis, and X-ray suggested perforation at the time of esophagoscopy. Mediastinitis and death resulted. No attempt was made to close the pin and efforts to push it into the stomach to turn it were unsuccessful.

9. After-care in foreign body cases should be given equal importance with operative procedure. Croup tent, forced fluids (often by vein or subcutaneously), oxygen direct with mask or by oxygen tent are often life-saving procedures. Tracheotomy if indicated and then proper care of the tube to insure sufficient airway is of utmost importance. Suction through the canula with adrenalin or salt solution dropped into it should be routinely carried out, especially if respiration is at all embarrassed. Foreign body cases should have the same attention we give our cases of tracheobronchitis after tracheotomy. We know that the drying of secretions in the canula, and lower in the trachea and bronchi, often is much more dangerous to the well-being of the infant than the infectious organism itself. As mentioned above, secondary bronchoscopy may be indicated and life-saving to remove secretions or an overlooked foreign body, especially where no tracheotomy has been done or seems necessary.

10. Overlooked foreign bodies present the real problem of bronchoscopy and esophagoscopy today. The diagnosis and treatment in this special field have advanced to a high degree of efficiency as a result of the untiring work of Dr. Chevalier Jackson and others. However, the great problem remains of getting information to the general medical men that foreign bodies in the air and food passages are much more common than they suspect, and that in any obscure chest condition they should be considered early rather than late after serious and sometimes fatal complications have arisen.

408 DAVIDSON BLDG.

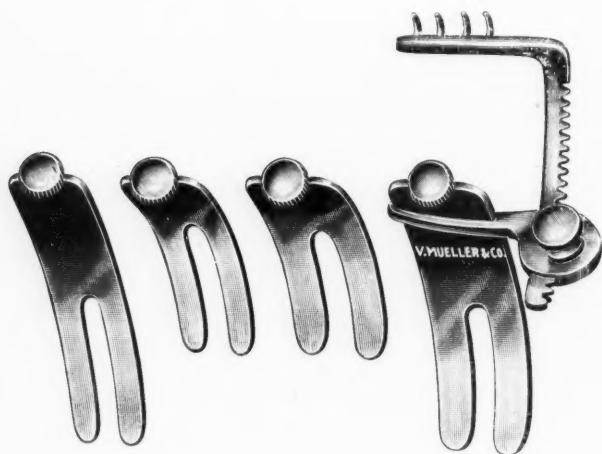
XXI.

THREE NEW INSTRUMENTS FOR THE FRONTO-ETHMO-SPHENOID OPERATION.

FERRIS SMITH, M. D.,

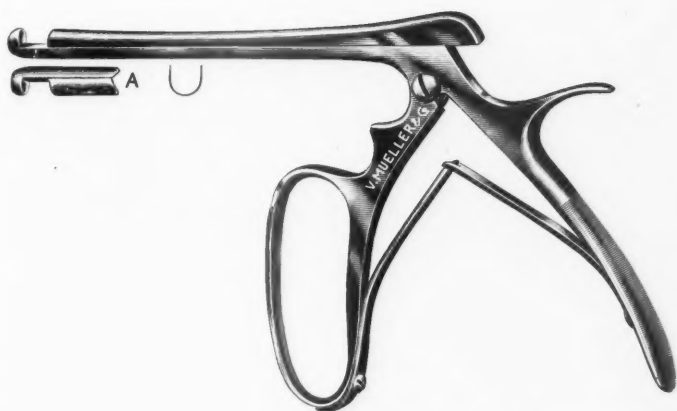
GRAND RAPIDS, MICH.

The following described instruments materially expedite and simplify the radical fronto-ethmo-sphenoid operation:



The self-retaining speculum (Fig. 1) is made right and left. The curved fingers on the fixed blade engage the skin and soft parts of the nasal side of the incision, while the movable portion, carrying a proper sized retractor blade, holds the periorbital away from the lamina papyracea so as to afford an excellent view and ample working space.

The blades are slotted to permit insertion over the posterior ethmoid artery, which puts this vessel on tension and facilitates its ligation. The blades are made in two lengths and widths.



The Kerrison type forceps (Fig. 2) are made "up and down." They are of very sturdy construction and have sufficient length to permit their use in removing the anterior sphenoid wall. The handles provide ease of manipulation and maximum strength in removing thick bone. They are particularly useful in removing the posterior margin of the nasal process of the maxilla and the floor of the infundibulum and frontal sinus.



The heavy punch forceps (Fig. 3) is designed for use through the nose in removing the sphenoid floor. This bone is frequently so thick that a forceps of great strength is required for its removal. A forceps of this strength is frequently required for section of the lamina papyracea in cases of long continued bone inflammation.

XXII.

THE EAR MICROSCOPE: ITS DESCRIPTION AND ITS USE AS A CLINICAL INSTRUMENT.

ALLEN B. POTTER, M. D.,*

ST. LOUIS.

The ear microscope is a relatively new instrument in the hands of otologists. On account of this fact, and because of the extremely limited literature available concerning the apparatus, it is my intention to give a detailed description of it and to set forth some of the results obtained in the use of it.

Various methods have been used in the past for more minute observation of the tympanic membrane and its related structures; these have fallen short of the desired results because of limited illumination and magnification.

In 1872, Mach and Kessel designed an instrument, in their laboratory in Vienna, for the purpose of magnifying the eardrum. This was not used extensively and was soon found to be impractical.

In 1923, Waar-produced a similar magnifying device by which he was able to see the blood stream in the vessels of the eardrum. But Lüscher describes this as "impracticable and a physiological rather than a clinical contribution."¹

Other instruments have been used, but with a limited degree of satisfaction, among them are the ophthalmoscope and the otoscope.

In 1924, Eckert and Mobius brought forth an ear telescope of 9-21 X magnification. This was an improvement over previous similar devices, but, since it was hand-operated, the otologist was at a decided disadvantage, and was unable to see details of the blood stream as was desired.

During the summer of 1925 Dr. E. Lüscher, of the Department of Otolaryngology, University Clinic, Bern, designed the instru-

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ment that is known as the ear microscope, and with which we are at the present concerned. (Fig. 1.)

The ear microscope has been in use in the Department of Otolaryngology of Washington University School of Medicine and Barnes Hospital for a little more than a year, and until recently was the only instrument of its kind in the United States. We have, during that year, been able to study for the first time the microscopic anatomy, physiology and pathology of the eardrum and its related structures in the living.

The only literature that may be found pertaining to the ear microscope and its uses has been produced by Dr. Lüscher, and since it is published in the German language, I shall quote in full his summaries:

ON THE TOPOGRAPHY AND PHYSIOLOGY OF THE SUPERFICIAL
CUTANEOUS VESSELS OF THE EARDRUM.²

1. The possibility of observing the eardrum vessels with the ear microscope permits an exact description of the topography and the superficial vessel network as well as the course of the blood stream in living humans.

2. The cutis layer of the eardrum contains, directly beneath the epithelium (connecting three dimensions), a widespread network of capillaries and veins that courses over the eardrum in various arrangement and depth. On the edge it hangs directly together with the corresponding superficial venous plexus of the outer auditory canal.

3. In spite of various draining possibilities there is a definite normal regulation of the blood flow out of the eardrum. The middle third of the *pars tensa* gives its blood toward the umbo and from there along the handle of the malleus over the cutaneous strips to the upper ear canal wall; the two outer thirds send their blood toward the periphery. The *pars flaccida* has its own irregular stream. The flow of blood follows in the deeper layers and normally is not to be seen.

4. The superficial network of vessels permits a division anatomically as well as functionally into three divisions:

- (a) The central vessel network of the *pars tensa*.
- (b) The peripheral vessel network of the *pars tensa*.
- (c) The vessel network of the *pars flaccida*.

5. The eardrum is a very adaptable object for estimation of the capillary pressure. This carries in the smallest capillaries an average of 60 mm. of water with extremes of 32 and 94 mm. water. In somewhat wider vessels (ca. 40 microns) the average is 94 mm. water with extremes of 56 and 136 mm. water. The first values are the lowest that can be observed in man.

6. Normally the majority of eardrum vessels are in tonic contraction and are closed to the blood stream so that with the maximum dilatation

the blood stream course is restricted to a very few vessels. With maximum dilatation the width of the stream course can rise to the value of twenty times and the rapidity of volume to 100-200 times.

ON THE FUNCTIONS OF THE STAPEDIUS MUSCLE IN HUMANS.³

1. The tendon of the stapedius muscle was observed directly with the ear microscope with strong magnification through a nonirritated defect in a drum of an ear otherwise normal.

2. The stapedius muscle of man shows regularly and constantly appearing cochlear reflexes, which can be set up equally from the same and the opposite ear with about the same intensity.

3. With pure tones the reflex appears only in a certain tonal range. This lay, for an examined individual, between 90 and 14,000 vibrations.

4. The tone must have definite intensity depending on the height. With rising intensity there first appears beginning jerks, finally it is a constant contraction which first disappears with cessation of the tones.

5. Noises set up the reflex much more easily than pure tones. Ordinary conversation 50 cm. distance, likewise soft noise of the breath at the ear, light tapping of the tuning fork, etc., is sufficient to bring about a jerking. The reflex seems, therefore, especially sensitive to those stimuli which impinge themselves upon the ear in daily life. It is so sensitive that in normal daily life it must be in constant activity.

6. The stronger, higher, and noisier the sound impression becomes, the more easily the reflex appears.

7. The setting up of the reflex is caused by mere anticipation of a strong sound stimulus. However, the expectation to a certain degree must be restricted reflexively, and cannot be brought about through listening.

8. The reflex can be released through slight stimulation; for an example, through pricking of the external ear.

9. Even with the strongest voluntary contraction of the muscles innervated by the facial nerve, no simultaneous contraction of the stapedius occurs. A voluntary contraction of the stapedius cannot be established.

10. The contraction of the stapedius seems to be isometric and no movement of the drum seems to follow.

11. All facts point to the conclusion that the contraction of the stapedius muscle causes a decrease of the sound transmission and thereby a decrease in hearing. From this viewpoint it is to be looked upon not as an agency for hearing but as a protective arrangement which has the function in a reflective automatic manner to protect the auditory perception apparatus from strong sound effects. The corresponding animal experiments of Kato were confirmed in the above description of the human ear examination.

ON THE OCCURRENCE OF SMALL DRY DEFECTS.⁴

The *pars flaccida* was searched for small open defects by means of the ear microscope with large magnification. With this, small dry macroscopically appearing perforating defects (Foramina Rivini) appeared as minute atrophies. An open defect was never found and therefore must at least

occur much less often than we supposed up to this time. It seems that minute atrophies were often spoken of as perforations. Only by examining with large magnification can a true distinction between atrophy and defect be made.

The microscope consists essentially of two parts:

1. A vertical light chamber, adjustable in itself, but firmly fixed to
2. A horizontal microscope barrel.

These two parts are so placed on the adjustable stand that they may be rotated freely in a horizontal plane and limitedly in a vertical plane.

The light chamber consists mainly of two cylinders, the larger one being at the top and containing a specially designed light, which may be shifted somewhat eccentrically to the lower or smaller cylinder by means of two thumbscrews. This shifting governs, to a certain extent, the direction of the ray of light down the barrel and changes its point of reflection from the mirror at the bottom of the cylinder. This ray of reflected light must, depending upon the magnification and the size of the ear speculum used, travel in a plane parallel to the line of vision through the microscope barrel. At the upper part of the smaller cylinder and just below the light is a lens which is of the same size as the lumen of the cylinder and which has a focal distance sufficient to concentrate the light rays on the adjustable mirror below. Again, and just below the lens, is a flat metal plate which contains several apertures, of different sizes, to govern the amount of light passed down to the reflector below. Immediately beneath this is a second metal plate with two large circular apertures, one of which contains a colored glass filter for eliminating the red rays, while the other is left open for a white light. The colored light is used in making special observations. Midway down the shaft of the smaller cylinder is a screw arrangement for lengthening the barrel and increasing the distance between the light and the mirror below.

The microscope barrel, as stated before, is stationary in relation to the light chamber, but the two together may be swung around 180 degrees on the base in a horizontal plane; they may also be tilted up and down in the vertical plane, the point of attachment

of the upper part of the stand with the mid-point of the microscope barrel being used as a fulcrum. The lens in the light end of the barrel is fixed and is directly below the reflecting mirror. The power of magnification is controlled entirely by the eye-piece used. There are six in number; one is plain or of very slight magnification, on which is etched a scale, in millimeters and fractions thereof, for measuring the field of vision; the others are numbered 5, 10, 15, 20 and 25, respectively, representing 10, 20, 30, 40, 50 X magnification. With the high magnification it is impossible to see but a small portion of the drum at a time (the relation between the magnification of this eye-piece and that of the plain is comparable to that between the oil and the low power in an ordinary microscope).

The stand is so constructed that the shaft may be raised or lowered several inches. The base of the stand is movable on a connected platform, and can be shifted forward, backward, and to right or left. The amount of this excursion can be very accurately noted by use of the three screws, two of which are calibrated. This is important in determining the distance moved to bring the different fields into focus.

The description given above is of the microscope proper, and, while stationary as compared to the easily manipulated aural speculum, it can be very quickly adjusted before and during examination to all desired positions.

For the purpose of convenience, a special table or tripod has been designed. It consists, as seen in the photograph (Plate 1), of a platform swung from one side of an upright standard, counterbalanced by a heavy weight. This platform turns freely in a horizontal plane, and can be raised or lowered by adjustment of the standard. Fixed to the platform and beneath it is a rheostat for controlling the intensity of the light. Lying on the floor is a second rheostat, which we have added, and which we find convenient when increased illumination is desired.

The examinations heretofore have been made with the patient lying flat on the back on a table, with his head extended over the edge and securely placed on the headrest. Recently, however, we have been making the examinations with the patient in a half

sitting position, in an ordinary ear, nose and throat chair. After some experience the operator will be able promptly to adjust the microscope to the proper height and inclination, and slight movement of the patient will not necessitate a general readjustment.

It is to be assumed that the operator has carefully prepared the external ear for examination by removing all cerumen or other obstructing material. The ear speculum should be black inside, as the reflected light from a bright one is confusing. After placing the speculum well down into the canal, the ear microscope can be adjusted. It is always well to examine with the low power first, as one would do with the ordinary microscope. The field of vision is inverted.

The tympanic membrane is very sensitive to all stimuli and to any changes taking place in related structures. Normally it is almost entirely void of blood supply; even the larger of its superficial vessels appear as fine glistening fiber strands, at times joined or interlaced and in a state of contraction. But as the speculum is gently moved against the canal wall, especially in the region of the isthmus, these fine threads take on a reddish hue and their identity becomes more evident. The larger vessels first begin to fill—and can be noted in the process of filling—then the finer vessels make their appearance and are seen to connect the larger ones.

The most abundant blood supply is seen to come in by way of the posterior fold, carefully avoiding the flaccid membrane. The portion of the tympanic membrane just below the posterior fold is next in order and has a rather abundant blood supply as compared to the anterior fold. The vessels entering by way of the anterior fold and posterior fold seem to recognize the handle of the malleus as the dividing line, each supplying blood to its respective part of the drum as it follows down the handle of the malleus, and only occasionally extending across. At the umbo these vessels interlace freely and send branches radially to the inner one-half or two-thirds of the drum. If irritation is continued or intensified, the peripheral vessels, which are somewhat more noticeable along the upper margin, are seen to fill and connect with those which have entered by way of the anterior and

posterior folds. The filling of these vessels, after repeated observations, impresses one because of its orderly arrangement. I have watched these vessels dilate, in the same and in different individuals, using various types of irritation, such as scratching the canal wall or introducing a gentle current of air; and if these sources of irritation are not applied too enthusiastically I have been able to anticipate the filling of certain vessels in order.

I have repeatedly introduced the speculum, using the lower magnification, and tried to get a dilatation of the superficial vessels in immediate relation to that part of the canal stimulated, but it seems as though the vessels entering by way of the posterior fold are on guard and are the first to make their appearance. From the foregoing, I would suggest that there is a very similar blood supply on the inner surface of the tympanic membrane, and that the connecting vessels between these two circulations play only a minor part except in the extremes. To me the circle of anastomosis around the umbo is quite interesting and fairly well defined. This will not be described until further observations have been made.

We have all seen bulging eardrums, made the usual incisions, watched the process of healing, and later viewed with delight the healed drumhead with all the landmarks present. But how many of us have analyzed our entire actions and procedures in the matter? Paracentesis has become too commonplace. We disregard our technic and rely upon the wonderful healing power of the membrane. Paracentesis should not be treated lightly. Every move made and every instrument used has a purpose. The illumination should be perfect; the knife should be sharp; the speculum, which should be of the exact size to fit the canal, must be properly adjusted; and last, but not least, the incision must be carefully and deliberately placed.

I have become interested in watching, with the ear microscope, the healing after paracentesis, and I have found that the upper terminus of the incision should be limited; in other words, it should be started at a reasonable distance from the vessels, entering by way of the posterior fold and continued downward, backward, then forward in an arc of a circle to a point well below

the umbo. It will be found that this incision heals much more quickly from above downward than from below upward. The explanation for this is that the blood supply in the upper and posterior part is more abundant. Injury to these vessels will embarrass the healing power of the drum. We have all labored diligently with perforations in this region, and have seen them resent every method of treatment. Granting that the ingrowth of the squamous epithelium and the nature of the attic suppuration must be mentioned as factors in healing, we must also consider the injury done to these posterior vessels. (Fig. 5.)

The only way to govern the upper limit of the incision is to start at that point, making the incision from above down. The drum membrane heals, following this type of myringotomy, with the minimum amount of scar. In two cases I examined there was a thickened band 1 mm. in width, passing from the periphery of the drum downward and forward to the malleus below the short process. This band was found to be from $1\frac{1}{2}$ to 2 mm. below but parallel to the posterior fold and contained all the blood vessels which normally pass in along the posterior fold. (Fig 2.) It is in this type of case that the upper limit of a high incision would cut off all the blood supply which normally courses down the posterior part of the handle of the malleus. This anomaly is another reason why the upper limit of the incision should be guarded. I doubt if we are ever justified in extending the incision upward in order to get additional drainage.

We are all familiar with the characteristic manner in which calcareous deposits are arranged on the drum. Never have I seen this forming, either in the upper anterior or posterior quadrants or around the umbo; but beginning always in the lower quadrant—more frequently in the anterior inferior than in the posterior—usually in a crescentic arrangement with the convexity upward and at a reasonable distance from the periphery and the umbo. In other words, the calcareous deposits very clearly map out that portion of the drum which is most deficient in blood supply; and the sluggish circulation in this part is explanation enough for its occurrence. The presence of rounded ends on this deposit also clearly shows its tendency to invade first that portion of the drum which is most deficient in blood supply.

SUMMARY.

1. The ear microscope was designed and first used by Dr. E. Lüscher, of Bern University, in the summer of 1925.
2. It can be very easily and quickly adjusted by an experienced operator.
3. It is the only means at the present time of making accurate microscopic examination in studying the anatomy, physiology and pathology of the ear.
4. The blood vessels of the tympanic membrane, free from pathology, are quite constant in their general arrangement and distribution.
5. The group of vessels entering by way of the posterior fold may course across the upper posterior quadrant of the drum unusually low.
6. Paracentesis should be done with care, the incision accurately placed to avoid injury to the vessels entering by way of the posterior fold.
7. The incision should be from above downward.
8. Incision well placed heals more quickly from above downward than from below upward.
9. Calcareous deposits are found in that portion of the drum which has the most sluggish blood supply.

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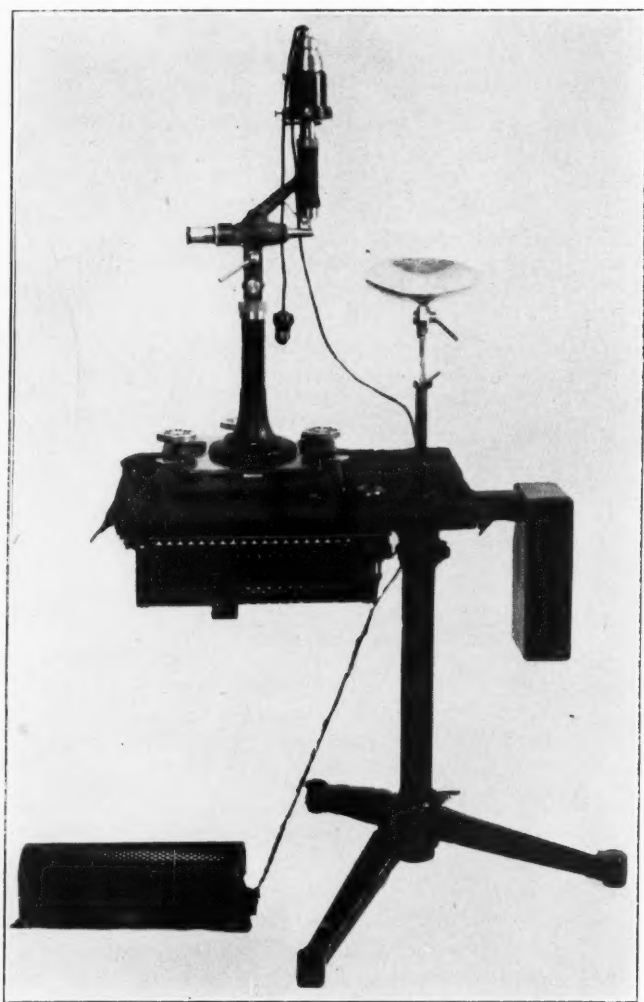


Fig. 1. The ear microscope, placed on the specially designed table or tripod.

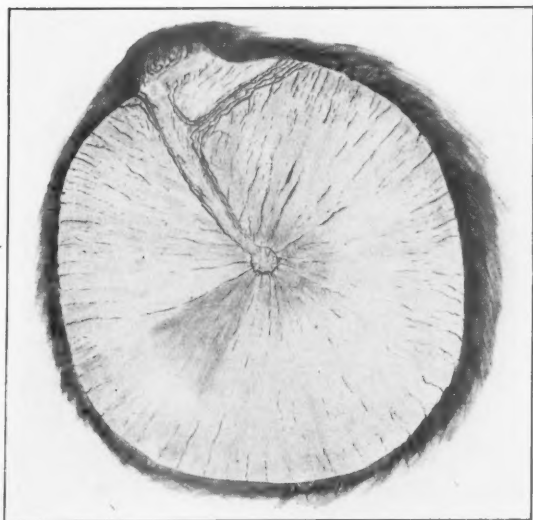


Fig. 2. The tympanic membrane, left. This was examined for the purpose of studying the superficial cutaneous vessels, and when viewed with the low power eye piece, appeared free from pathology or defects. But when the circulation was stimulated by irritation of the external canal, and the low power eye piece was replaced by one of higher magnification, it was seen that the entire blood supply which normally enters along the posterior fold was confined to a small thickened band parallel to but considerably below its usual location. Two rather large vessels were seen to pass upward toward the short process while the remaining vessels passed downward along the handle of the malleus to the umbo. The image, when seen through the ear microscope, is inverted.

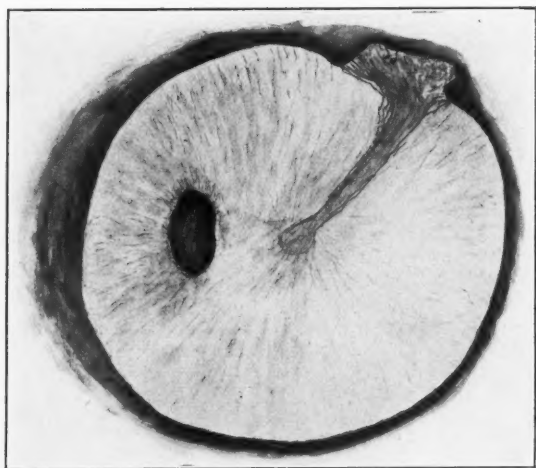


Fig. 3. The tympanic membrane, right. Patient aged 20. Ten days after paracentesis. The drum is somewhat thickened. The incision healed very readily, more quickly from above downward than from below upward. Even with the high power magnification no scarring could be seen; no microscopic change in the circulation was evident.

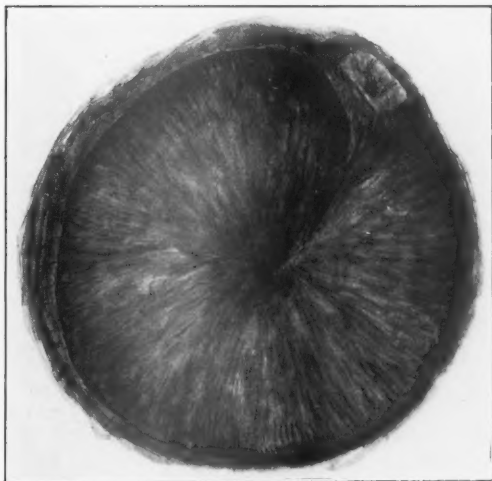


Fig. 4. An acutely inflamed and bulging eardrum as seen with the use of No. 15 eye piece.



Fig. 5. The tympanic membrane, right. Patient age 18. Otitis media, duration three years. The drum is quite thick, especially in the upper posterior quadrant. The vessels, which normally extend downward along the posterior surface of the handle of the malleus and umbo, have been destroyed with some embarrassment to those along the posterior fold. A polyp is seen through the upper part of the large perforation, lying in close contact with the incus. High up in the posterior part is a mass of unhealthy granulation tissue completely covering over the stapedius muscle.

XXIII.

THE PRACTICAL VALUE OF THE AUDIOMETER.

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This paper has to do only with the individual (not the group) audiometer and is based entirely on the use of the 2-A audiometer. The 2-A audiometer produces eight pure tones, ranging from 64 to 8192 double vibrations per second in octave steps. It is possible to increase and decrease the intensity of each tone to the threshold of hearing of a patient and then record it on a graphic chart or audiogram. This chart may be filed as a permanent record of the individual's hearing at that time, also it is possible to figure the percentage loss of hearing. Only the regular telephone type of receiver was used. The value of the bone conduction receiver may be the subject for a further presentation; however, it seems of little practical importance at this time.

The feasibility of evaluating methods or procedures in clinical medicine depends upon many factors, chief of which is the individual's thorough knowledge of and long experience with other procedures and methods which the one under consideration is to replace or complement. Before one expresses an opinion on the value of the audiometer he should have had a thorough-going interest in and an extensive experience with the other methods of testing hearing, especially with tuning forks and whistles of accepted value. It has long been my opinion that nothing less than the forks and whistles devised by Bezold were sufficient for satisfactory work. Also, it is essential that patients have been subjected to audiometric study supplemented by the other standardized methods.

Hearing tests may be divided into quantitative and qualitative.

QUANTITATIVE TESTS.

1. Spoken Voice.—Because of the importance of hearing the spoken voice in human contact, this is in the majority of indi-

viduals the most important test of hearing. One may have lost completely the low or high tones, but if the middle of the scale is retained near normal he may have noticed no difficulty in hearing. While the spoken voice is the one thing the average patient wishes to hear, it is as a test of hearing dependent entirely on the individual examiner, and I am certain that even the same examiner will vary the intensity of his speech from day to day or even minute to minute. It is not overstating that the spoken voice test can be interpreted only that at the time the test is made the patient is or is not able to hear the examiner at a certain distance.

2. Forced Whisper.—This has some advantage over the spoken voice as a hearing test, because it is of value from the qualitative as well as quantitative standpoint. The probability of day to day constancy of intensity of the forced whisper is greater than is true with the spoken voice, also, the intensity of different examiners is more likely than with the spoken voice. The forced whisper has some qualitative value; for example, the number forty-four is of rather low pitch and sixty-six or sixty-seven of high pitch.

3. Watch Test.—This test is of little value unless the same watch is used at every examination; also, because the beat of the watch gives only one or at best a mixture of only a few tones.

4. Acoumeter.—This test has the same advantages and disadvantages as the watch.

5. Audiometer.—This will be considered later.

QUALITATIVE TESTS.

1. Tuning Forks and Whistles.—Tuning forks and whistles, as devised by Bezold, are the only type which should be considered. These have a range of from 16 d. v. to about 30,000 d. v.; however, the method is rather crude and cumbersome. There is no satisfactory method of standardizing the stimulation of the fork and certainly no method of recording with any accuracy the hearing of the patient for various tones. Also, because of the practicability of using the upper tones only by air conduction it

is quite impossible to eliminate one ear in the test. The tuning forks have a distinct advantage over the audiometer in testing the lower portion of the tone scale, but are very unsatisfactory in the upper portion.

Under tuning fork tests one must mention the three outstanding ones which are of great practical value in ear examinations—i. e., Weber, Rinne and Schwabach. These tests are of the utmost importance in diagnosing the location of lesions in the hearing apparatus; however, while the question asked by Hetrick, "Are we as sure of our fork tests as we think we are?" may seem like heresy, there are at times conflicting findings in examinations which justify a very critical study of accepted methods and ideas.

To briefly illustrate the justice of a complete re-examination of accepted methods, I might mention a type of finding which is not at all infrequent—i. e., the patient with impacted cerumen in one ear only with the Weber (made with A fork) lateralized to the poorer ear, the Rinne (made with a fork) plus in the affected ear, the Schwabach (made with the a fork) prolonged, which would localize the defect in the conducting apparatus but who shows on more careful tests to have a lesion in the apparatus of perception, also, this type of finding is more easily demonstrated with the audiometer than by any other means.

This character of finding should cause one to accept without argument the apparent fact that fork tests and the audiogram are both of great importance and that they are neither one sufficient unto themselves but should complement one another.

THE AUDIOMETER.

The audiometer may be considered an apparatus for both quantitative and qualitative examination.

As a quantitative test it is of great value because it enables the otologist to secure an accurate record of hearing, which may be compared with other graphs made on the patient at any subsequent period. This is especially useful in watching the progress of a patient while under treatment. Without some such definite

method one can never know with any degree of accuracy the progress which a patient is making under treatment.

It is my custom in the average middle ear lesion to make a careful audiogram and after four to six treatments to make another for comparison.

This is the only method with which I am acquainted whereby one may with reasonable certainty check the statement of the patient about progress, for if he is an optimist he always thinks he improves for a time with any treatment, while if a pessimist there is no improvement; the audiogram is the court of last resort.

A point of great importance is the similarity of audiograms made by different persons on the same patient. One should standardize the method of procedure, after which it is possible to have the graph made by a nurse or anyone who understands what is to be done. This can be done rapidly and with a minimum of error and becomes a most valuable part of the patient's record.

The audiogram is the only method by which otologists at a distance can compare findings on a given patient.

In making tuning fork tests the patient must be of a rather high intelligence to disregard what he wants to hear and what he actually hears. In this regard the audiometer is very valuable, because the patient does not try to answer what he thinks he should hear but only what he hears. Young children can frequently give a very satisfactory audiogram, while they rarely are able to answer the questions in fork tests.

A word should be said regarding the acceptance of the percentage loss of hearing as shown by the audiogram and the actual loss to the individual; this is especially true in industrial cases. While the percentage loss of hearing may be very accurate, it probably does not denote accurately the practical loss which the patient has sustained. As an example, the high tones might be entirely destroyed and yet if the middle of the scale is retained perfectly serviceable hearing be present.

It has been previously mentioned that the audiometer was the most satisfactory method of testing the upper portion of the scale, and it is here that it is of great clinical importance. With forks

and whistles it is practically impossible to exclude one ear when testing the upper range, while with the telephonic receiver of the audiometer it is very frequent that the patient fails with one ear when the receiver is in contact to hear the upper tones, but as soon as he takes it away from his ear he hears the tone with the other ear. When there is a drop in upper tones a diagnosis of a perception apparatus lesion is justified. Drury³ calls especial attention to the importance of the drops in 4096 d. v. (C5), even with recovery of the 8192 c. v. (C6) in early lues. This he calls the dipper curve, and I can substantiate his statements regarding the importance of the drop in 4096 d. v. However, frequently in my cases I have found the 8192 d. v. absent.

The use of the audiometer can, I believe, give one an entirely new conception of the management of certain types of hearing defects. While one might get the same information with the fork tests, it could never be so graphically and definitely called to his attention. As a point of practicability, I think this is one of the outstanding features of the audiometric method of testing hearing. Drury³ says: "The audiometer is the instrument of choice in diagnosing early involvement of the auditory nerve." This opinion is substantiated by Shambaugh.¹⁷ When a drop in the upper tones is found one certainly should examine that patient in the same manner that he should be examined if he had an optic neuritis. As Drury³ says: "The recognition of syphilitic auditory neuritis is of paramount importance, as it may be evidence of an incipient cerebrospinal involvement."

Not only syphilis should be considered, but if the absence of syphilis can be demonstrated the patient should be examined for other general conditions, such as a focus of infection which might and occasionally does cause perception apparatus lesions. In other words, one should have the same conception of the cause of perception apparatus defects as of nerve lesions in any other part of the body. While these defects are not as successfully treated as in some other nerve lesions, improvement is obtained sufficiently to make it justifiable to be ever on the alert for this type of trouble.

When one considers the practical value of the audiometer, he is immediately faced with the opinion of Shambaugh,¹⁷ who

says, "The audiometer is a mechanism of some value for studying the physiology of hearing, but so far has been shown to be of very little practical value to an otologist." This opinion is probably well founded, for a man so competent to make and interpret other hearing tests as is Dr. Shambaugh, but I am certain he knows, as I know, how few men will give the time necessary to make a satisfactory examination with forks and whistles.

It has been almost twenty years since I read a paper before the Section on Eye, Ear, Nose and Throat of the Illinois State Medical Society, on "The Subjective Tests of Hearing," and every little while now someone asks for a reprint of that article. However, I am convinced that a very small proportion of otologists are equipped, mentally and physically, to make satisfactory fork tests. For some reason, many otherwise competent men are not willing to procure or use forks and whistles which are acceptable for good work.

CONCLUSIONS.

The audiometer is a practical mechanism for making graphic records of hearing by which the future progress of the patient may be judged.

The audiometer is the only mechanism by which a reasonably accurate percentage of hearing loss may be determined.

The audiogram is the only practical graph by which a patient may be watched by different otologists.

A satisfactory audiogram can be made in many patients who seem unable to co-operate sufficiently for good fork tests. This is especially true in children.

There are other methods superior to the audiometer for testing the lower tones.

The audiometer is the best mechanism for testing the upper portion of the scale.

Defects in the upper portion of the tone scale call for a careful general examination of the patient.

An audiometric study should be made in every patient who complains of defective hearing.

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XXIV.

THE EAR IN HEAD INJURIES: PRACTICAL CONSIDERATIONS.

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It is the purpose of the writer to discuss the subject of this paper in as practical a fashion as possible, avoiding theoretical and scientific discussions of the subject matter. For this reason no particular reference to authorities will be made. Anyone desiring to go into the theoretical and scientific end of this subject may do so by consulting other published papers. I have used the word "head injury" advisedly. I wish to avoid the use of the word concussion as much as possible, inasmuch as "concussion" in the minds of many individuals means a period of unconsciousness. The history of unconsciousness can be elicited in many of the cases exhibiting ear symptoms after a head injury, but unconsciousness is not necessarily an important factor. Ear symptoms may appear in the head injury case without the history of unconsciousness. In other words, in as far as the damage to the ear mechanism is concerned, we cannot divide head injuries into severe injuries and minor injuries.

Pure concussion results in a group of symptoms which are the result of a temporary inhibition of one or more of the medullary centers and which are not accompanied by pathologic lesions. Pure concussion does not mean the death of the cell, but it does mean a cell paresis, and from this paresis a complete restoration to function should take place. Most of the cases which we will examine, however, are the result of concussion, plus contusion, plus petechial hemorrhages, following which there are inflammatory reactions and processes of repair which leave sequelæ of a more or less permanent character. In the examination of these cases we must forget that we are pure otologists and remember that the brain, as well as the ear, is damaged in practically all cases. For the above mentioned reasons I do not like to use the term "concussion" in a discussion of these injuries.

Character of the Injury.—Most of the injuries with which we have to deal are caused by a broadly acting force, as the result of which the head receives a blow or the head may strike an object, or the head may suddenly be jarred against the vertebral column, as in a fall from a height, or the head may be caught and compressed by two large objects one which is in motion.

The Mechanics of the Injury.—The mechanics of the injury may be studied in three distinct phases.

Phase 1.—At the moment of impact the hard skull strikes against the brain at the site of the impact, because of the fact that the brain, which is floating free in its cerebrospinal fluid, does not quickly take up the motion of the skull.

Phase 2.—The brain is taken along with the skull in the direction of the motion of the skull.

Phase 3.—The side movement of the skull is suddenly stopped by its own vertebra or against some solid object, and it comes to rest. The brain is still moving toward this point of rest and strikes the inner surface of the skull at this point.

Injury to the Skull.—An injury as outlined in the preceding paragraph may or may not fracture the skull. This point is really immaterial to the otologist, because he is mainly interested in the actual injury to the brain itself if the patient survives. A fracture of the skull is usually *prima facie* evidence that the brain has been injured, but is not interesting to us on its own account. The theory of skull fractures is based upon the elastic properties of the skull. It is easy for us to conceive that the infant skull with its large open fontanels is elastic, but we must also remember that the adult skull possesses and retains some of these elastic properties. Fractures are divided into bending and bursting types. A small force applied to the vault of the skull produces a bending fracture in its immediate neighborhood. A larger and more severe force, acting over a larger area of the skull, produces a bursting type of fracture. The skull is compressed in the direction of the force and is spread at right angles to this direction. The vault represents an arch of uniform thickness. The base of the skull is divided into three fossæ, separated by stronger portions of bone. On account of the large number of foramina, certain portions of the base are weak and

other portions are strong. Because of these distinct differences in the structure of the vault and the base, the greatest number of bursting fractures are found at the base of the skull. According to various authors, from 60 to 80 per cent of all skull fractures are fractures of the base. Of the entire base the middle fossa is the weakest portion, and for this reason the middle fossa suffers more frequently from fractures of the base than do the anterior and posterior fossæ. In fractures of the base the temporal bone is very frequently injured. Many of these injuries result in death, while in others the patients survive and we examine them later. Even if the temporal bone is not actually fractured it is certainly set into heavy swinging movements and its contents are thrown into violent motion and commotion.

Temporal Bone Fractures.—Temporal bone fractures may be present in very many different forms, but for otologists they assume in the main two general patterns: first, the longitudinal fracture, which is by far the more frequent; and second, the transverse fracture.

Longitudinal Fractures.—Longitudinal fractures are usually found along the anterior border of the pyramid, beginning somewhere in the region of the Gasserian ganglion, extending along the anterior border of the pyramid and ending in the tegmen antri or tegmen tympani. They may also end in the external canal. This type of fracture may, by fracturing the margo tympani and by extension into the external canal cause hemorrhage into the external canal. If this occurs, it is then a definite evidence of a basal but not necessarily of a labyrinthine fracture. This type of fracture may occasionally send small offshoots into the labyrinth, but it may also cause hemorrhage into the labyrinth without actually fracturing it.

Transverse Fractures of the Pyramid.—The transverse fractures of the pyramid are far less common than the longitudinal type but are just as typical in their course. They cross the pyramid at right angles to its long axis at its weakest portion, which is the region of the vestibule. They destroy all the membranous portions of the inner ear, both vestibular and cochlear. They only rarely involve the lateral wall of the inner ear and enter

the tympanic cavity, but when they do they produce a rupture of the drum with hemorrhage into the external canal or a hemato-tympanum with an intact drum. When they do so enter the middle ear the prognosis of the case must be extremely guarded, because of the danger of subsequent infection from the middle ear with a resulting meningitis and death.

Practically all fractures of the middle fossa and the temporal bone must be diagnosed clinically, because of the fact that the X-ray is notoriously incompetent to delineate these fractures.

Injury to the Brain.—Injury to the brain or the skull contents may be considered under four general heads:

First, the actual injury to the brain tissue itself;

Second, trauma to the blood vessels and their regulating mechanism;

Third, hemorrhage by direct laceration of larger blood vessels; and,

Fourth, the tearing and laceration of cranial nerves.

Number 1. Injury to the Brain.—Through compression of the skull by the force exerted against its exterior, the brain is compressed. The lateral ventricles in turn are compressed by the brain tissue and tend to collapse. The spinal fluid is forced from them through the foramina of Monro into the third ventricle and from there through the aqueduct of Sylvius into the fourth ventricle. Inasmuch as the fluid cannot escape from the fourth ventricle, there takes place here a whirl-pool action of fluid under pressure. The main force of this whirl-pool action is exhibited against the walls of the fourth ventricle and especially against its floor. When we recall the location of the cochlear and vestibular nuclei, immediately beneath the surface of the floor of the fourth ventricle, we can see with what ease these structures may be injured by this pressure wave of fluid with resulting contusions and petechial hemorrhages in this territory.

Number 2. Trauma to the Blood Vessels and Their Regulating Mechanism.—In addition to the actual damage to the brain tissue by pressure, we must consider trauma to the blood vessels and particularly to their regulating mechanism by contusion together with the resulting inflammatory changes and processes of

repair. It has been shown by a number of observers working with experimental animals that a traumatic paralysis of the vasoconstrictor nerves takes place following a head injury. This has been thought to be due to some trauma in the midbrain. Ricker was able to show that after trauma there were areas of brain in which there was present a condition of stasis and pre-stasis. In these areas the vasoconstrictors are paralyzed or non-irritable. The end result of this condition is an interruption of tissue function and tissue necrosis. Phelps also called attention to the effect of a blow on the vasomotor center, causing vasomotor depression of the cerebral vessels with paralytic dilatation of capillaries, stasis, thrombosis and punctate extravasations, the effects of which were increased by the edema of the brain. In relation to our work as otologists or as neuro-otologists, I certainly wish to stress the point that this paralysis of vasoconstrictor nerves takes place in the central vestibular area at the floor of the fourth ventricle as well as in other portions of the brain. It has also been shown by observers that this process does not tend to cease but may be found months and years after the original injury, and this probably explains why the symptoms which we shall discuss later are present years after the resulting head injury.

Number 3. Hemorrhage by Laceration of Larger Blood Vessels.—Hemorrhage by direct laceration of blood vessels does not concern us much as otologists.

Number 4. Tearing and Laceration of Cranial Nerves.—The tearing and laceration of cranial nerves is caused by the movement of the brain within the skull. In this connection it is found that nerves are injured by stretching in inverse proportion to their length. The short ones are frequently injured and the long nerves are very infrequently injured. This accounts for the frequent injury of the eighth nerve.

INJURY TO THE EAR END ORGAN.

Injury to the actual end organ of hearing may result because of transverse fracture of the pyramid, because of longitudinal fracture of the pyramid, because of the tearing or stretching of the eighth nerve, and finally because of an injury to the end organ by a wave motion of fluid.

The force which is exerted against the outside of the skull compressing its contents and setting the brain in motion is also exerted against the temporal bone. It is inconceivable that this force should be communicated to the inner ear structures through the hard temporal bone, but this compression of the brain against the temporal bone can compress two soft structures which are connected with the inner ear. These structures are the region of the internal auditory meatus and the sacculus endolymphaticus. Pressure on these two structures produces a wave motion of perilymph and endolymph within the inner ear. The energy of this wave motion expends itself in the region of the round window, which is the weakest portion of the bony inner ear. This accounts for hemorrhages in this neighborhood, for bulging of the round window membrane into the middle ear and for its occasional rupture. For the substantiation of this statement one must study the pathology of human beings who have succumbed to head injuries and also the pathology of experimental animals subjected to head injuries. These facts can be ascertained from the published literature and will not be entered into in this brief discussion.

CLASSIFICATION OF HEAD INJURIES FROM AN OTOLOGIC
STANDPOINT.

- I. Injury of the brain with vestibular symptoms, but no deafness.
- II. Injury of the brain with injury of the inner ear and deafness.
- III. Injury of the brain and of the inner ear by temporal bone fracture.
- IV. Injury of the eighth nerve.

SYMPTOMATOLOGY: HISTORY TAKING.

1. The age of the patient. This is very important with reference to the prognosis of the case, the prognosis for a partial or complete restoration to function being much better in the young than in the old.
2. The Present Complaint. We try to allow the patient to develop his complaint in his own words without leading questions.

Most of these patients have a neurotic overlay and it is easy to suggest symptoms to them which will then become a part of their symptom complex.

3. The Time of Injury. This is important, because it is necessary to know the length of time which has elapsed between the injury and the time of examination, in making a prognosis.

4. The Memory of Events Immediately Subsequent to the Injury. We never ask a patient if he was rendered unconscious by his injury because most of these patients are looking for compensation and regard a period of unconsciousness as important. However, if a patient can tell you clearly what took place immediately after he was hurt and can describe in detail how he was hurt, we know that he was not unconscious. I do not lay too much stress on this history of unconsciousness, because I have discovered that the sequelæ of a head injury may be produced without a period of unconsciousness.

5. Bleeding from the Nose, Mouth or Ears. This information is sometimes elicited from the patient and sometimes from the attending physician. The importance of this point is for the diagnosis of skull fractures. Bleeding from the nose or mouth in the absence of other symptoms is indicative of a fracture of the anterior fossa, while bleeding from the ears is indicative of a fracture of the middle fossa.

6. We next inquire when the symptoms of headache, vertigo, tinnitus or deafness first appeared, and we inquire about the character of the same; whether the tinnitus was continuous or intermittent; whether the vertigo was continuous or intermittent, and if intermittent how long the spells of vertigo lasted.

7. Vertigo and its Nature. There is nothing so distressing or disabling to the patient who has recovered from a head injury as the vertigo of which he complains. It is absolutely wrong for us to dismiss this patient lightly and say that his vertigo is due to a neurosis. We must examine him very carefully and try to determine whether or not his vertigo is of a true labyrinthine character and if it was caused by the injury sustained.

Spontaneous labyrinthine vertigo is divided into:

1. Turning vertigo;

2. Tactile vertigo. Tactile vertigo is again divided into:

- a. Errors of sensation; and
- b. Lateropulsion.

1. Turning or rotatory vertigo is the *most certain sign of vestibular invasion, peripheral or central*. There is nothing else in the brain which can produce it. The patient who complains of turning vertigo states that external objects move in a circle in relation to himself, or that he seems to move in a circle in relation to external objects. As a rule, the movement of the external objects is more frequently complained of, and this movement is always in the horizontal plane. It does not particularly matter whether the objects move or the patient moves; the essential thing is that something moves and moves in a circle. *If a patient exhibits true turning vertigo he must have or must have had spontaneous vestibular nystagmus.*

2. Tactile Vertigo. Much less is known about tactile vertigo. When suffering from tactile vertigo the patient feels as though the bed were tilting or the foot end of the bed were rising; as if the ceiling were coming down; as if the side of the chair on which he is sitting is falling. He feels uncertain on his feet; he feels as though he were drunk; he complains of things going black before his eyes. While these errors of sensation are not as important as the true turning vertigo, they are definitely not hysterical. They may be definitely designated as vestibular in character, but they are not as a rule accompanied by nystagmus. They may represent an earlier stage of turning vertigo. These errors of sensation are in character if they are accompanied by caloric hyperirritability. This point will be taken up later in the examination of the patient.

In lateropulsion the patient feels as though he were pushed or pulled laterally to one side; never forward or backward. These attacks may be mild or they may be severe. They may at times be severe enough to simulate epilepsy. They are found in non-suppurative diseases of the central vestibular system, and therefore they may be found as the result of head injuries which involve the central vestibular area. They are not nearly as frequent as turning vertigo or errors of sensation.

The factors which influence or produce vertigo are: (1) rolling over in bed, (2) sitting up suddenly, (3) looking up suddenly,

(4) bending the head forward or backward suddenly, (5) going up above ground level, (6) undue exposure to sunlight, (7) even the mild use of intoxicants, and (8) undue physical or mental exertion.

8. General Neurological Symptoms. The patient may complain of nervousness, sleeplessness, irritability, depression, anxiety, loss of memory, etc.

9. The kind of occupation engaged in. This is important, because we will be obliged to determine whether the individual can return to it.

10. We take a history from the patient whether any work has been attempted since his injury and what kind of work he has engaged in, and we do this in order to help us in estimating the severity of the symptoms and later in computing the amount of the loss of function.

EXAMINATION OF THE PATIENT.

1. The need of teamwork. For the diagnosis of the case and the care of the individual who has had a head injury, the services of the surgeon, internist, neurologist and otologist will be needed. In the immediate care of the patient, the services of the surgeon are most important. In the later examination of the patient for the purpose of making a diagnosis and a prognosis, the services of the otologist are of prime importance. Even at this time he will need the cooperation of the ophthalmologist, the neurologist and the internist.

2. The general examination of the patient is divided into:

A. *The immediate examination*, which is made as soon after the injury as possible; and

B. *Subsequent examinations*, which are made when the patient is again up and about and which may be necessary years and decades after the original injury.

A. THE IMMEDIATE EXAMINATION: It is hard to tell how many head injuries develop ear symptoms, because of the fact that they are first seen by the surgeon or the general physician. They are only examined by the otologist at a later period, and then only when the physician or surgeon suspects some ear lesion. However, regarding this point Mygind, at the Copenhagen General

Hospital, examining all head injuries brought into the hospital, whether minor or major, found otoscopic changes in 50 out of 142 cases, and traumatic vestibular disease in $33\frac{1}{3}$ per cent. Even this does not represent the true percentage of ear defects after head injuries, because not all head injuries are brought into the hospital.

1. *Otoscopic Examination:* We examine for the presence or absence of fresh or dried blood in the external canal; for perforations of the drum; for fracture lines in the bony external canal wall; for redness of the posterior superior canal wall and the drum adjacent thereto. Occasionally cerebrospinal fluid is found in the canal. If blood or cerebrospinal fluid or a hematoma is found on the first examination sterile cotton is placed in the ear and no further examination of the ear is made until we are satisfied that the drum has surely closed. The nurses and internes are instructed to regard this ear as a *nolle me tangere*. This is insisted upon because of the danger of infection and meningitis from manipulation of this ear.

2. *Rough Hearing Tests:* The hearing tests which are made at this period can not be exact, because we are making them in hospital rooms or homes where conditions are unsatisfactory, and because of the fact that in many instances the patient's sensorium is blunted at this time.

3. *The Presence or Absence of Spontaneous Vestibular Nystagmus.*

4. *Inequality of the Pupils and Their Reflexes.*

B. SUBSEQUENT OTOLOGICAL EXAMINATIONS:

1. *Eyes:*

a. Pupils: The pupils are examined to see whether they are round, whether they are equal or unequal and whether they respond to light and accommodation.

b. Muscles: Rough tests are made to see whether there are any muscle paralyses.

c. Media and Fundi: Media and fundi are examined.

d. The vision is taken at 20 feet and at 14 inches. If the vision is below normal at 20 feet or 14 inches it may be necessary to call

in an ophthalmologist to determine whether this loss of vision is due to a refractive error or to some condition which has nothing to do with the accident sustained.

2. *Otoscopic Examination:* The external canals and the drums are again examined in the same fashion as that outlined under the immediate examination.

3. *Hearing:* The following examination is made to determine the cochlear function.

a. *Whisper:* The voice examination is made because it has been a standard method of examining the ears for years. It has its disadvantages. It is difficult to whisper with a uniform force. The small size of the usual examining room is another disadvantage. The patient is confused by the reflexion of sound from the walls and the ceiling of the room. My method is as follows: Each ear is tested separately with the eyes closed, the opposite ear also being closed. The patient is placed at the opposite end of a room twenty feet long. The ear to be examined is turned away from the examiner. The examiner is turned away from the patient. If in these positions the patient hears the whisper of the examiner we feel that he has a fairly normal hearing for the whisper.

b. *The watch:* This is tested for with one ear closed and both eyes closed and the same watch is used in all cases. We do not of course recommend the watch as a standard way of testing hearing, but inasmuch as it is commonly used by patients and has been used by otologists for many years we include it in our examination.

c. *The C. 5 Fork:* We test each ear separately with the C. 5 fork to determine the upper limits, and for this purpose we also use the Galton whistle.

d. *The Rinne Reaction:* This is tested for on each ear separately with a fork of 256 double vibrations using the upper portion of the mastoid. In most cases with a traumatic deafness, if not too pronounced, we will find that we have a relatively lengthened positive Rinne, which is due to:

e. *Shortened Bone Conduction:* To determine the bone conduction we use the regulation Schwabach test. In the cases where there has been a traumatic involvement of the cochlear side of

the ear we usually find a shortened bone conduction which is definitely due to traumatic inner ear disease, causing a limitation of the upper tone range. This was present in a large majority of my 275 cases and when present there was always found an accompanying limitation of the upper tone range. In all examinations for bone conduction we must take great care to avoid the "fatigue" phenomenon. The Weber reaction is uniformly taken in these cases but has not been found of much use.

f. *Audiometer:* In these examinations the Western Electric No. 2A. Audiometer is used. In the examination of hearing the audiometer, of course, has its distinct advantages and its disadvantages. The advantages are that we can obtain a curve of the entire tone range in a relatively short time. This can also be done by a continuous series of forks, but takes a great deal of time and effort and usually results in a tiring of the patient. Another advantage is the possibility of rechecking the patient with sound of the same intensity at later periods. Another advantage is the fact that it is not possible for the patient to mislead quite as easily as with the voice or the watch test. The disadvantages of the audiometer have been pointed out by Shambaugh and others, and they are these: We must be careful to avoid bone conduction to the opposite ear. This disadvantage can somewhat be eliminated by instructing the patient to hold the ear piece of the audiometer to his ear lightly as he would a telephone receiver and not press it tightly against his skull. Furthermore, this objection to the audiometer holds only in the lower and the lower middle tone ranges. In the upper tone range the objection does not hold.

The traumatic loss of hearing which we usually encounter in these cases is a loss in the upper tone range, and when such loss in the upper tone range is encountered the prognosis for its recovery is bad. Another very common finding is a notch in the region of the C. 5 tone, the tone of 4096 double vibrations. When a loss of hearing occurs in the lower tone range and is definitely not due to disease in the conducting mechanism or to a fracture of the middle ear structures, we can assure the patient that that portion of the hearing curve will improve after the lapse of time. In regard to these special notches for the C. 5 tone above noted, care must be taken in all of these cases that the patient is free

from syphilis. It has been stated by Drury of Boston that syphilitic patients very frequently present this same tone notch in the region of the C. 5 tone.

4. *Spontaneous Vestibular Symptoms*.—The spontaneous vestibular symptoms are really more important in the examination of these patients than the examination of the hearing, because of the fact that the findings are entirely objective. The patient can neither produce them nor inhibit them. These spontaneous symptoms are nystagmus, falling reactions, pointing reactions, gait deviations and the girdle responses.

(a) *Nystagmus*:

1. *The Kind of Nystagmus*: We have to deal with osculatory and jerky nystagmus types. The osculatory is an even, to and fro movement of the eye. The jerky nystagmus consists of a slow component and a quick component. The slow component is the vestibular component and the quick return is the cerebral compensating act. None of the osculatory forms of nystagmus are labyrinthine in character. The jerky nystagmus, however, may be labyrinthine. Every labyrinthine nystagmus is jerky, but not every jerky nystagmus is labyrinthine.

2. *The Form of Nystagmus*: There are many forms of nystagmus, but the typical labyrinthine nystagmus has both horizontal and rotary components. In this connection we not infrequently see in normal patients a purely horizontal nystagmus in both end positions of the eyes, and these are usually equal in amount and degree.

3. *The Direction of the Nystagmus*: A nystagmus is called after the direction of its quick component. This is somewhat misleading, because, as we stated above, the quick component is a cerebral compensation act. The real vestibular portion of the nystagmus is the slow component, which is caused by the labyrinth. It is sometimes very difficult to tell in a very fine nystagmus whether it is osculatory or jerky, but if we look at some small blood vessel on the eye we will usually be able to tell in which direction the nystagmus goes, and if we can determine the direction of the nystagmus it is certainly jerky. When this does not suffice I would advise the use of an ophthalmoscope.

4. *The degree of the Nystagmus:* The degree of the nystagmus is usually designated I, II and III. If a nystagmus appears only on looking to the extreme right and not on looking straight ahead or to the left, it is a nystagmus of I degree toward the right. If a nystagmus appears both on looking to the right and on looking straight ahead but not to the left, it is a nystagmus of II degree; and if a nystagmus appears in all three positions of the eye it is a nystagmus of III degree. If a nystagmus does not increase when looking toward the quick component it is not labyrinthine in origin. We cannot draw many conclusions from the frequency and the amplitude of the nystagmus, though in general a very slow, coarse nystagmus is cerebellar in origin.

The Clinical Value of Jerky Nystagmus: In this connection we have two questions to answer: Question No. 1: Is the nystagmus vestibular or is it nonvestibular in origin? Question No. 2: If the nystagmus is vestibular in origin, is it peripheral or central in origin?

Question No. 1: Is the nystagmus vestibular or nonvestibular in origin?

1. Every vestibular nystagmus must be associated with vestibular vertigo (turning vertigo). Every nystagmus can be associated with dizziness but not necessarily with turning vertigo.

2. There must be an association of the eye movements in a vestibular nystagmus; if the eye movements are disassociated the nystagmus is not vestibular.

3. Abnormal forms of nystagmus:

(a) Diagonal nystagmus: This is always of nonvestibular origin.

(b) A retracting nystagmus (rare). This is due to a lesion in the Sylvian fissure and is always nonvestibular in origin.

(c) Vertical nystagmus: This may be vestibular or it may be nonvestibular, but it is usually nonvestibular in origin. We can only designate it as vestibular in origin if it is accompanied by turning vertigo, and when this is the case it is not of peripheral but of central origin. If this is true the lesion must lie in the territory of the anterior end of the spinal acoustic root, which lies in the region of the corpora quadrigemina. Therefore, if a

vertical nystagmus is accompanied by a vestibular vertigo the lesion is in the region of the corpora quadrigemina.

Question No. 2: Is the vestibular nystagmus central or peripheral in origin?

1. *Dizziness:* Both forms may be combined with dizziness.

(a) In a *peripheral vestibular nystagmus* there is a harmony between the intensity of the nystagmus and the intensity of the vertigo.

(b) In *central vestibular nystagmus* there is a disharmony between the intensity of the nystagmus and the intensity of the vertigo. We may have a large nystagmus and a small vertigo or vice versa.

2. *Falling reaction.*

(a) In *peripheral vestibular nystagmus* the patient usually falls in the direction of the slow component and the fall is dependent upon the position of the head.

(b) In *central vestibular nystagmus* the fall is in the direction of the quick component and is dependent upon the position of the head.

(c) Cerebellar falling is usually always in the same direction and has no connection with the existing nystagmus.

Central vestibular nystagmus is produced by an organic or vasomotor disturbance in the central vestibular area. Spontaneous nystagmus may not be present at every examination. One should search frequently for it and should not say that it is absent after only one examination. If we are in doubt whether a very fine nystagmus is present or not, we should use the ophthalmoscope. Occasionally if a spontaneous vestibular nystagmus is not present, it may be produced by the head movements test. In the head movements test the examiner takes the head of the patient between his two hands and tells the patient to relax. The head is then slowly moved in every direction and when thoroughly relaxed it is suddenly tilted back. At this point the patient is directed to look first to the right and then to the left, and very frequently a spontaneous nystagmus appears. From my examination of these cases we are not able to conclude which vestibular area is diseased from the direction of the spontaneous nystagmus.

B. *Falling Reaction*: The human body has three apparati for balance which I name in the order of their relative importance.

1. The sensibility of the body muscles, both superficial and deep;
2. The vestibular innervation of all the body muscles;
3. The eye and the field of vision.

We can maintain our normal balance and relationship in space with two of these apparati intact, but not with one alone. Therefore in the Rhomberg reaction we cut out the effect of the eyes, and we are then dealing with the deep and superficial sensibility of the body muscles and the effect of the labyrinthine motor innervation of the body muscles. In the Rhomberg reaction there is a normal tendency to sway slightly forward and backward, inasmuch as the human body is not as symmetrical from front to back as it is from side to side. However, any marked swaying forward and backward is apt to be hysterical. An actual falling backward is apt to be hysterical if it is protected and cerebellar in origin if it is not protected.

The labyrinth has two general effects or actions:

- (a) A sensory action in which it reports to the cerebellum the position of the head in space, and
- (b) A motor reaction in which it innervates all the muscles of the body.

MOTOR LABYRINTHINE FALLING REACTIONS.

1. In acute conditions of the labyrinth;
2. In acute conditions of the vestibular nerve;
3. In acute conditions of the central vestibular area;
4. In acute diseases of the cerebellum;
5. In cerebellar tumors.

1. *Acute Diseases of the Labyrinth*: In this condition the patient usually falls in the direction of the slow component of the nystagmus, and this reaction is often dependent upon the position of the head in the regular way—i. e., no matter what the position of the head in space, the patient falls in the direction of the slow component. In these acute conditions of the labyrinth the falling reaction may not always be found.

2. *Acute Conditions of the Vestibular Nerve:* We have the same general findings as in No. 1, but not so often dependent upon the position of the head.

3. *Acute Conditions of the Central Vestibular Area:* In this condition the patient, as a rule, falls in the direction of the quick component, and this reaction is dependent upon the position of the head in the regular way.

4 and 5. In these conditions the patient, as a rule, falls toward the diseased side, independent of the position of the head and independent of the existing nystagmus, unless the cerebellar lesion has a secondary pressure effect on the central vestibular area.

C. *Pointing reactions:*

1. We examine for this reaction only in the upper extremity. We can examine either with the shoulders up and have the patient point down toward the finger, or with the shoulders down and have him point up toward the finger. Of the two reactions the latter is probably the more accurate, inasmuch as fewer muscles are involved.

2. According to the directions of E. Lewis, we usually make this test in three positions: directly forward, slightly to the left and slightly to the right of the midline.

3. After having taken the actual pointing reactions we do a deviation reaction. The two hands of the patient are stretched forward; the eyes are closed; they touch the fingers of the examiner; the examiner then moves his fingers back but keeps them in the same relative position, and the patient is directed to hold his hands where they were. Sometimes one arm and sometimes both arms will deviate either to the left or to the right. This is a drift reaction.

The pointing act is a cerebral act governed by inward and outward pointing centers in the cerebellum. These centers in turn are capable of being influenced by stimuli coming from the two vestibular areas. (In this connection note the action of artificial stimulation of the labyrinth in the production of past pointing.) If a patient with an intact cerebellum has a past pointing reaction he has either (1) vertigo, or (2) disturbances in the motility of the arms or (3) both. To be of any value at all, the past pointing

must be three or four inches or more. We are not able to predict the site of the lesion from the direction of the past pointing. The pointing reactions of the lower extremities are unreliable. In this connection we use the forward and backward progression with eyes closed. This gives us a sort of a combined pointing and falling reaction of the whole body.

(5) *Functional tests of the labyrinth:*

(a) *Turning Tests.*—The examination of the patient in the turning chair has its advantages and its disadvantages. The advantage is that it stimulates only one single set of canals. The disadvantage is that it stimulates both labyrinths at once. We irritate only the set of canals which is at right angles to the axis of turning. With the head erect the horizontals are irritated. With the head ninety degrees forward or back the vertical canals are irritated. In this connection the theory of the endolymph movement toward and away from the ampulla must be borne in mind, and we must remember that the action of the flow toward the ampulla has twice the value of the action of the flow away from the ampulla.

(b) *Test for Vertigo:* The patient is turned to the right ten times in twenty seconds with eyes closed, and during the turning and after the turning he is asked to tell the operator whether he is turning and in which direction.

(c) *Test for Nystagmus:* The patient is turned ten times in twenty seconds with eyes closed, and after the turning is stopped he is directed to look at a distant object. The amount and time of the resulting nystagmus is then carefully noted.

(d) *Test for Past Pointing:* The patient is turned ten times in ten seconds, and the pointing reactions are then taken as in the spontaneous pointing test.

The above three reactions are taken with the patient in the sitting position and with his head erect.

(e) *Test for Falling:* The patient sits in the chair with his head in his lap, and the chair is very slowly turned five turns to the right. The patient is then directed to sit up and note is made as to whether he sits up toward the right or in a straight line or toward the left.

D. *Caloric tests:*

1. The fractional caloric method of Kobrak.
2. The mass douching of Bárány.

No. 1. The Kobrak Method.—This method is used mainly to test for hyperirritability or hypoirritability of one or both labyrinths and of one or both vestibular systems. The patient sits in the examining chair, head tipped forward thirty degrees and eyes in position I. Exactly 5 cc. of water at 60 degrees F. is injected into the right ear. The time of the beginning of the injection is noted and the eyes are then closely watched for the first appearance of nystagmus. This normally appears after a latent period of from fifteen to twenty-five seconds. This nystagmus normally lasts from sixty to ninety seconds after its first appearance. If the nystagmus begins in 10 seconds or less, we know that we are dealing with a hyperirritable end organ. If the resulting nystagmus lasts longer than two minutes we know that we are dealing with a hypersensitive central vestibular area. If 5 cc. of cold water does not produce any reaction we then test the ear with 10 cc. and subsequently with 20 cc. of water at 60 degrees C. If this produces no reaction we revert to the mass douching of Bárány. The advantages of the Kobrak method are that it does not produce vertigo, that it does not distress the patient and that it is as nearly as can be made qualitative in character. The disadvantages of the method are that it does not produce past pointing or falling.

No. 2. The Mass Douching of Bárány.—If we wish to study perverted pointing and falling reactions after cold water douching, we revert to the mass douching with water at 68 degrees F. and study the resulting nystagmus with the head forward thirty degrees and then back sixty degrees. At the same time we can study the pointing reactions with the head in both positions. Supposedly with the head forward thirty degrees we irritate only the vertical canals and with the head tipped back sixty degrees only the horizontal canals. It is, however, quite probable that we never irritate only one set of canals, and that with the head tipped forward thirty degrees we irritate mainly the vertical canals, but we do also get some effect from the horizontal canals. With the head tipped back sixty degrees we irritate mainly

the horizontal canals, but we do also get a slight action from the vertical canals. Therefore, the resulting nystagmus will never be absolutely and purely either horizontal or rotary, but the horizontal nystagmus will always have a slight rotary component, and the rotary nystagmus will always have a slight horizontal component.

In the foregoing, I have detailed briefly and somewhat sketchily my method of examining the ear in head injury cases. With the information which this examination furnishes I should be able to tell whether the ear mechanism has been damaged either peripherally or centrally. The estimation of the hearing defect should be a very simple matter. With respect to the vertigo, the matter is not quite so simple. We can definitely tell whether the vertigo is vestibular or nonvestibular. If it is vestibular, it is far more disabling than the loss of hearing.

The estimation of disability because of traumatic vertigo rests upon a number of different factors, such as the frequency of the spells of vertigo and their length of duration, the age of the patient, his occupation, and so forth. Even with these factors to guide us the estimation of the disability will always be more or less arbitrary guesswork, based upon a large experience.

XXV.

OBSTRUCTIVE LARYNGEAL DYSPPNEA.

A. N. CODD, M. D.,

SPOKANE.

Obstructive laryngeal dyspnea is not a specific disease. It is a symptom-complex, important in the diagnosis of various affections of the larynx.

The common conditions precipitating this symptom-complex are the acute edemas of the larynx. Simple acute laryngeal edema of inflammatory origin may arise in a child following a mild pharyngitis with scarcely any other symptoms. It also may complicate retropharyngeal abscess or any severe infection of the nose or throat. The invading organism in these cases may be the hemolytic streptococcus or the pneumococcus; the diphtheria bacillus plays its part and occasionally the cultures show a mixture of organisms.

Angioneurotic edema of the larynx is frequent, laryngismus stridulus occasional. Acute edema of the larynx has occurred in nephritis and in cases in which there has been a prolonged use of iodides. Angioneurotic edema of the larynx may occur in a highly nervous subject or may be the result of anaphylaxis. On direct examination of the larynx two large globular masses, of paler color than the normal mucosa, appear in the region of the arytenoids. If the cords can be seen they are thin and white, lacking the appearance of inflammation and edema that occurs in the inflammatory or nephritic type of edema.

Some of the chronic diseases, such as tuberculosis, syphilis and leprosy, and the neoplasms are not uncommon causes of obstructive laryngeal dyspnea. Other causes are abductor paralysis of the cords following thyroidectomy, various wounds of the larynx and irritating gases. This condition sometimes occurs following prolonged bronchoscopies or the prolonged retention of vegetable

foreign bodies. It develops, as a rule, eight or ten hours after instrumentation and should be anticipated.

The diagnosis of this condition is readily made by the indrawing observed at the suprasternal notch and at the ensiform cartilage during inspiration. Stridor often may be heard upon entering the room. Advanced cases show marked restlessness and pallor. Cyanosis is not the rule.

The prognosis of obstructive laryngeal dyspnea is in direct proportion to its recognition. Unrecognized cases die if complete obstruction occurs. There should be no mortality in a recognized case of complete obstruction if proper treatment is given immediately. Statistics show that about 33 per cent of the cases of angioneurotic edema of the larynx die of asphyxiation, obviously for the want of a prompt tracheotomy.

Treatment: In early cases of obstructive laryngeal dyspnea a regime of utmost vigilance should be followed; absolute rest, including vocal rest, is necessary. Sprays of cocain and adrenalin are very useful. Irritating swabs and applications are dangerous. Sedatives, such as morphine, are absolutely contraindicated. They produce listless asphyxia and prevent the slightest fight or struggle for air that characterizes acute asphyxia. Multiple incisions in the edematous area by direct laryngoscopy have proven very beneficial, particularly in adults.

In advanced cases there is only one treatment. It is a low tracheotomy. Quoting Dr. Chevalier Jackson:

"There are only three exceptions to this rule:

1. If the case is one of laryngeal diphtheria and there is a skilled intubator who will not only introduce the intubation tube but will remain in the house to reintubate in case the tube is expelled, intubation is worth consideration; otherwise it is not.
2. Laryngismus stridulus is accompanied by indrawing, but the dyspnea may subside without tracheotomy.
3. Retropharyngeal abscess may require only evacuation."

CASE REPORTS.

Case No. 1.—Child, 16 months old, with following history: The patient had been ill two days with a slightly sore throat and a temperature of 99° and suddenly developed difficult breathing. Two hours after the onset

of this difficulty the family physician was called. He came immediately. On his arrival a frantic mother informed him that her child had stopped breathing.

Examination revealed the child in a state of listless asphyxia with the heart still beating. There was no cyanosis but the child was very pale. The doctor turned the child over and as he did so it took a struggled breath and started to breathe. A few moments' observation revealed a marked indrawing at the suprasternal notch and a feeble stridor. The child was taken to a hospital where I saw it a short time later. The indrawing was now very marked, both at the suprasternal notch and at the ensiform. The stridor was fairly loud and pallor was present. It seemed that each breath would be the last. A tracheotomy, using novocain anesthesia, was performed and a size two Jackson trachea cannula was inserted. Breathing immediately became normal. The indrawing and stridor ceased and the pallor subsided. For two days the temperature remained at 99 to 100 degrees. Throat and trachea cultures revealed a mixed growth of staphylococci and streptococci. No membrane was observed at any time, but direct laryngoscopy showed generalized edema of the larynx. Three days later the child developed an intense purulent bronchitis and had much difficulty in getting up the secretions. In order to overcome this complication intratracheal aspirations, two to five daily, were necessary. A eustachian tube catheter was inserted into a bronchus through the tracheal fistula and suction applied. Following each aspiration dyspnea ceased and breathing was quiet until the tracheobronchial tree filled up again. The child was too weak to expel its own secretions and would have drowned in them had it not been for the aspirations. This procedure continued for a week and required the most diligent observation by capable nurses. Aspirations were then discontinued and the trachea tube removed. Direct laryngoscopy revealed a normal larynx and the child made a complete recovery.

Case No. 2.—The patient was a woman, age 25, giving a history of a sore throat with fever ranging from 99 to 102 degrees for three days. Breathing became labored on the fourth morning, and by evening she showed signs of urgent laryngeal dyspnea. The tonsils and pharynx were highly injected. No membrane was present, but there was considerable edema in the aryepiglottic folds and over the cords. The glottic chink was practically obliterated. The stridor was marked. There was some pallor and considerable indrawing at the suprasternal and supraclavicular spaces. Cultures from the throat showed a pure growth of hemolytic streptococci. Cold adrenalin and weak cocain solution sprays were used without success. Multiple incisions were made in the edematous folds by direct laryngoscopy, using a Jackson laryngeal knife. The urgent dyspnea was relieved and the symptoms subsided within a few hours. Three months later a tonsillectomy was performed and cultures from the crypts were hemolytic streptococci.

Case No. 3.—This patient was a girl, age 6, with a history of hoarseness for five days. Difficult breathing then developed. Throat cultures by the family physician were negative for diphtheria but showed a mixed infection. On examination, five hours following the onset of the dyspnea, there was a very marked indrawing at the suprasternal and supraclavicular

notches and at the ensiform. Stridor could be heard upon entering the room. The child was very pale. Mirror examination of the throat showed generalized edema of the larynx. Tracheotomy was immediately performed, using a size three Jackson tube. The symptoms subsided immediately and the child fell asleep from utter exhaustion produced by this difficult type of breathing. Three days later the swelling subsided and the tracheotomy tube was removed.

Case No. 4.—The patient was a child, age 2. A peanut kernel was lodged in the right main bronchus, having been there six weeks. The foreign body was removed by bronchoscopy. Twelve hours later typical symptoms of obstructive laryngeal dyspnea occurred and became so urgent that low tracheotomy had to be performed. Normal breathing was resumed. After a few days the edema subsided and the cannula was removed.

Case No. 5.—The patient was an adult male of highly nervous temperament. He gave a history of a "nervous breakdown" six months previously. At that time he was hoarse for three weeks. The present illness dated back three days with an onset of hoarseness. At the end of the third day he developed difficult breathing and hysteria. Upon examination I found a moderate indrawing at the suprasternal notch with some stridor. He was restless but I attributed it to his nervousness. Mirror examination of the larynx showed two globular masses of edematous mucosa presenting at the base of the tongue. These did not completely obliterate the airway of the larynx. They were paler than the usual swellings of acute infection. The vocal cords were thin and not affected. I sprayed his throat with a 2 per cent cocain and adrenalin mixture and moved him to a hospital. The indrawing became worse, so I made several incisions into the globular masses by direct laryngoscopy. The urgent symptoms subsided but a moderate swelling persisted for ten days and then disappeared.

Case No. 6. Chronic Laryngeal Stenosis.—The patient was a woman, age 38. There was a history of choking and wheezing at frequent intervals for 12 years following an attack of diphtheria. Her speech had been more or less a hoarse whisper during the last five or six years. She had been diagnosed and treated for asthma without relief. Choking attacks had been nearly fatal on several occasions. The condition of the present illness showed difficult breathing of three days' duration accompanying a severe sore throat and cold. The morning of the fourth day dyspnea became very marked. Examination revealed an intensely injected pharynx and larynx, a very narrow and fixed glottic chink, a slightly perceptible mobility of the arytenoids, heavy, dense adhesions crossing at the base of the vocal cords and extending midway toward the anterior commissure. There was a slight swelling of the cords, quite sufficient to cause almost complete obliteration of the airway. The diagnosis: Acute laryngitis superimposed upon a chronic laryngeal stenosis. Steam inhalations and sprays had no effect upon this condition and with the onset of marked indrawing at the suprasternal notch, with stridor and restlessness, a low tracheotomy was performed. The symptoms of obstruction subsided. Two weeks later a left ventriculocordectomy by direct laryngoscopy was performed to overcome the chronic laryngeal stenosis. In another week the tracheotomy tube

was removed. Examination of this patient two years following the operation showed good mobility of the arytenoids, a wide glottic chink but with some adhesions present. The voice is very much improved; speaks aloud easily, and there have been no more choking attacks.

PAULSEN MEDICAL AND DENTAL BLDG.

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A—Inspiration:

B—Expiration.

Fig. 1. The marked indrawing at the suprasternal notch and ensiform on inspiration is diagnostic of obstructive laryngeal dyspnea.



Fig. 2. Three views of angioneurotic edema of the larynx in the same patient at different times. There is lacking the translucent, bluish, watery appearance of the inflammatory type of edema.

XXVI.

POSTOPERATIVE USE OF RADIUM IN NASAL POLYPS.*

HORACE R. LYONS, M. D.,

CHICAGO.

Nasal polyps have been known for many decades and described for at least eighty years, yet the etiology and treatment, so far, have been a fruitful field for investigation and discussion.

The etiology of nasal polyps is generally believed to have as a necessary foundation the presence of chronic sinus disease, although the latter may be of a suppurative or a hyperplastic type. In its earliest stage the sinus membrane shows all the typical changes of chronic inflammation, namely, first: thickening of the membrane with infiltration by leucocytes, chiefly of the lymphocytic type; second, there is marked edema of the stroma through which is found lymphocytes, plasma cells and frequently eosinophiles; third, in the vascular system is found phlebitis and lymphangitis; fourth, osteomyelitis and periostitis of the underlying bone is the rule. With this picture of the pathology of chronic inflammatory disease, we have the basis for polyp formation. As the pressure of edema of the tissue continues, arteries, veins, lymphatics, elastic and fibrous tissue become less pronounced and more widely separated. This turbulence continues and finally produces a mass of tissue surrounded by a very thin layer of flat epithelium and containing edematous stroma in which there are no normal histologic structures. In this tissue, pressure and degeneration have eliminated fibrous, glandular, elastic and muscular tissue. Arteries and veins have largely disappeared. The adjacent bone and periosteum show various pathologic stages of inflammation. This may represent the formation of a myxomatous polyp.

*Read January 17, 1931, before the sectional meeting of the Laryngological, Rhinological and Otological Society, Ann Arbor, Mich.

In a contribution, ten years ago, the writer divided polyps into four types having pathology as the basis for the classification. First, myxoma, which is the pale grapelike tumor and microscopically has widely separated stroma by edema, throughout which glandular and fibrous tissue are not found. Secondly is the adenomyxoma, which is the myxoma described above, plus the addition, microscopically, of glandular structures. These two tumors, as found in gross specimens in the nose, have no characteristics which lead to differentiation and are as of one type when considered for postoperative treatment by radium. Also, these two types make up the great majority of polyps as found in the nose. Thirdly is the fibromyxoma which again is, microscopically, the myxoma with the addition of fibrous tissue in varying amounts. Grossly this tumor is often more firm than the myxoma and clinically is far more satisfactory to deal with. Fourth is the fibroma. This tumor presents the gross picture of a more solid tumor; microscopically the edema of the myxoma is replaced by fibrous tissue, and clinically this polyp is usually cured by complete surgical removal. It is therefore evident that the more fibrous tissue a polyp contains the better is the prognosis as to ultimate cure.

It was with this clinical fact in mind, plus the microscopic evidence that radium stimulates the production of fibrous tissue, that the writer, in 1919, started to use radium postoperatively in nasal polyps and first published its use in 1921.

In general, the treatment of these tumors presents two problems, first, the elimination of the underlying sinus pathology, and secondly, the introduction of some means to reduce the rapidity of recurrence of the polyp. The postoperative use of radium for nasal polyps was introduced to combat the latter of these two problems.

While this paper does not consider the first of these, namely, the elimination of the underlying sinus pathology, yet the proper use of radium and its results depend entirely upon the success of the operative procedure. It is therefore evident, from the voluminous discussions of the treatment of chronic sinusitis plus the frequent practical inaccessibility of the disease, that nasal

polyps will continue to recur until this problem is solved. These facts, however, do not eliminate any form of treatment directed toward lengthening the period of recurrence of the polyp tissue. In the treatment of this condition, following thorough operative measures, radium is inserted into the polyp area to stimulate the production of fibrous tissue in the membrane which tends to recur as polyps. Microscopically, it has been shown that by the postoperative use of radium the original polyp, which was a myxoma or adenomyxoma, recurs containing fibrous tissue. Also, the more fibrous tissue produced in the recurring tumor by radium the greater is the length of time before nasal obstruction demands another operation. It follows also that the removal of a myxoma into which has been introduced fibrous tissue by radium, that subsequent radium treatments tend to further delay its recurrence.

The postoperative use of radium for nasal polyps introduced this element into a new field where the dose became important, especially so because radium burns were reported in its use in other tissues. Early, a dose of fifty milligram hours was used. This has been increased and for the past seven years 400 milligram hours has been introduced forty-eight hours after operation and repeated on the eighth and fifteenth postoperative days. In no instance has a burn or necrosis developed, and the amount of local reaction and swelling has in no case been important. The radium, a fifty milligram tube with attached string, sealed to the cheek by adhesive tape, is inserted into the polyp area and packed tightly in place forty-eight hours after operation and allowed to remain eight hours. During such treatment no discomfort is experienced.

The use of radium, for thirteen years, in these cases has produced several rather definite conclusions. In the first place, radium introduced into the substance of a polyp will have no effect upon the tumor. This misconception early had the effect that some rhinologists found no benefit in the use of radium. Secondly, the proper use of radium for this condition is a method which delays but does not eliminate the recurrence of polyps following removal. Whether or not the polyp recurs depends upon,

first, the accessibility of the pathology; second, the extent of sinusitis; and third, the thoroughness with which the polyp and its underlying pathology is or can be removed.

While radium was not introduced to cure polyps nor to prevent their recurrence, it has proved to be of substantial importance in the treatment of those cases which have been carefully operated upon, by lengthening the interval of freedom from obstruction and by making subsequent operations more encouraging.

30 No. MICHIGAN AVENUE.

XXVII.

TYMPANOGENOUS PURULENT LABYRINTHITIS: REPORT OF CASES.

FREDERIC G. SPROWL, M. D.,

SPOKANE.

The invasion of the inner ear in tympanogenous labyrinthitis may occur in one of several ways, namely, (1) through the round window, (2) the oval window or (3) by circumscribed erosion of the bony capsule.

The usual route in acute purulent labyrinthitis is through the windows, and of the two the round window is the most frequently attacked, this being the weakest point in the bony capsule. As pointed out by McKenzie,¹ several authors, especially Hayman, have confirmed this by animal experimentation. Uffenorde claims that in twenty-nine cases of acute purulent labyrinthitis the invasion occurred twenty-seven times through the windows. In circumscribed labyrinthitis Ruttin² believes the horizontal semi-circular canal is the most frequent site of the erosion, reporting thirty-one fistulae in fifty cases.

McKenzie³ speaks of a "window diffusion" serofibrinous labyrinthitis resulting from dialysis of bacterial toxins through the round window without rupture of the secondary membrane. This is based upon the animal experimentations of Uffenorde, who claims that under certain conditions the serofibrinous type may change to the purulent form without actual rupture of the membrane. Ruttin accepts this view. Usually, however, there is destruction of this membrane, and the infection in the tympanic cavity comes into direct communication with the inner ear. Fremmel⁴ is of the opinion that purulent labyrinthitis is preceded by a serous labyrinthitis and that the transition is so rapid in many cases that it is unrecognizable. The resistance of the patient and the virulence of the organism probably determine the interval of time between the two.

Purulent labyrinthitis from capsular erosion occurs in the same manner but is slower in developing. A circumscribed labyrinth-

itis may exist for years as an irritative lesion and then undergo complete healing. Cases of this type are frequently discovered during the performance of a radical mastoidectomy, and complete healing may follow, with or without restoration of function. Lowered resistance or the presence of a virulent organism, however, may change this lesion into the diffuse purulent type. Cholesteatoma and tuberculosis play an important rôle in the capsular erosion type, both showing a slow, progressive bone destroying tendency.

Clinically we must differentiate between

1. Circumscribed labyrinthitis.
2. Diffuse serous labyrinthitis.
3. Diffuse purulent latent labyrinthitis.
4. Diffuse purulent manifest labyrinthitis.

In circumscribed labyrinthitis a patient with chronic purulent otitis media complains of occasional attacks of headache, tinnitus, and not infrequently vertigo. The hearing is impaired but not completely lost, and the turning and caloric tests reveal some vestibular function. There may or may not be a positive fistula test. As long as the inner ear reacts to stimulation (vestibular or cochlear), labyrinthectomy is contraindicated, a radical mastoidectomy usually sufficing to clear up the condition. If necessary the labyrinth can be drained at a subsequent time.

Diffuse serous labyrinthitis is usually sudden in onset. The patient complains of severe vertigo with nausea and vomiting. There is a nystagmus toward the healthy side, and the hearing markedly reduced or absent. In most cases, however, there is some response to the caloric test. If a positive fistula test existed before the onset it can usually be elicited during the attack, even in the absence of a response to caloric stimulation.

In diffuse latent labyrinthitis the function of both the cochlea and vestibular mechanism is completely destroyed, but active symptoms of labyrinthitis are absent. The cerebrospinal fluid should be normal. In the presence of a healed chronic purulent otitis operation is unnecessary. If active purulent otitis and mastoiditis are found, mastoidectomy with labyrinthine drainage is indicated. Many surgeons who delay inner ear drainage unless visible evidence of labyrinthine necrosis is found will disagree

with this assertion. The fact remains, however, that the first evidence of activity is an extension to the meninges, and labyrinthectomy performed with this complication carries a high mortality. Drainage of the labyrinth before the infection reaches the posterior fossa involves no more risk than a radical mastoid operation.

Diffuse purulent manifest labyrinthitis is, as a rule, easily recognized. Nystagmus (third degree), vertigo, nausea, vomiting, past pointing and falling, with a dead labyrinth and deaf ear, make the diagnosis comparatively easy. The cerebrospinal fluid may show an increased cell count with serous or purulent meningitis. Whatever the fluid findings, immediate labyrinthectomy is indicated. According to Fremmel,⁵ this procedure has been followed in the Neumann Clinic for several years and has materially reduced the mortality. He admits that serous labyrinthitis sometimes gives the above picture, and if not operated on will frequently recover with some restoration of function, but the necessary delay for a differential diagnosis may result in a fulminating meningitis. If diffuse suppurative meningitis is present with bacteria in the cerebrospinal fluid, not only should the labyrinth be drained by the Hinsberg or Neumann method but an opening through the cochlea to the spinal fluid should be made.

Fremmel also believes that stapedialexation during a radical mastoidectomy is so frequently followed by a diffuse suppurative labyrinthitis and a fatal meningitis that this accident calls for immediate drainage of the labyrinth.

The importance of the spinal fluid examination should not be overlooked in purulent labyrinthitis. Certainly any suspected invasion of the endocranium calls for an immediate spinal tap. Many authors hold that eight or ten white cells to the field is indicative of meningeal irritation. Ruttin⁶ believes this should be raised to forty or fifty. Polymorphonuclear cells are an abnormal finding and their presence an indication of a suppurative process in or near the spinal fluid. The presence of globulin is pathologic, and when found in excess should suggest meningeal irritation. Kopetzky⁷ holds that the absence or diminution of the copper reducing substance in the spinal fluid is probably the earliest sign of beginning meningitis.

Repeated spinal taps should be resorted to if for any reason operative interference is delayed in diffuse manifest labyrinthitis. By this means, and this means only, can early extension to the meninges be detected.

SUMMARY.

1. Labyrinthine drainage is contraindicated as long as a vestige of cochlear or vestibular function remains.

2. In circumscribed labyrinthitis the radical mastoid operation will usually suffice.

3. In diffuse latent labyrinthitis inner ear drainage should be combined with the radical mastoidectomy.

4. Immediate labyrinthectomy is imperative in diffuse manifest suppurative labyrinthitis. Should the cerebrospinal fluid disclose the presence of bacteria an opening through the cochlea into the spinal fluid should be made.

5. Either the Hinsberg or Neumann operation meets the surgical requirements. The former is technically less difficult, while with the latter there is less danger of injuring the facial nerve.

Case 1.—Female, age 40. O. M. P. A., left, three and a half weeks. Postinfluenzal. Facial paralysis twenty-four hours' duration. Left temporal headache. Small central perforation with sagging posterior superior quadrant of drum. Immediate simple mastoidectomy. Cortex sclerosed with granulations in the antrum. Findings suggested O. M. P. C., but patient denied previous ear history. Discharged from hospital ten days later. Facial greatly improved, but complained of some vertigo. No nystagmus or headache. Noise apparatus in healthy ear disclosed a total deafness. No reaction to caloric stimulation. Ten turns to the right produced no nystagmus. Ten turns to the left produced a nystagmus to the right lasting ten seconds. Patient insisted upon returning home. Six weeks later severe frontal headache, nausea, vomiting, and rigidity of neck. Spinal fluid cloudy; 3,000 polymorphonuclears to the cu. mm. No organisms. Radical mastoidectomy. Small area of necrotic bone on the prominence of the horizontal canal. Curettement was followed by a gush of pus. Hinsberg operation. Eleven days later left the hospital. Complained of headache and vertigo for several weeks. Cavity dry in

six weeks. Eighteen months later ten turns to the right produced a nystagmus to the left lasting eleven seconds. Ten turns to the left produced a nystagmus to the right lasting eighteen seconds.

Case 2.—Male, age 7. O. M. P. A., left ear, three weeks. Acute nephritis, probably scarlatinal, of two months' duration, with albumin and blood in urine. Red blood count, 2,200,000. Hemoglobin, 38 per cent. Subperiosteal swelling. Simple mastoidectomy revealed flabby granulations and free pus. Culture showed pneumococcus. Small area of sinus exposed at operation, and appeared healthy. Nine days later facial paralysis and rotary nystagmus toward healthy ear (second degree). Hearing markedly impaired. Moderate vertigo. Swelling and tenderness over left mandibular articulation, closing the auditory canal. Two days later hearing completely gone with noise apparatus. Nystagmus continued, but no nausea, vomiting or past pointing. Twelve days after first operation large quantity of pus evacuated from zygomatic area far forward. Area of dura exposed in middle fossa found congested. Given a transfusion on the table. Two days later severe headache, rigidity of neck and Kernig. Spinal fluid cloudy; pressure 44 with 960 cells; organism, pneumococcus. Twenty cc. of patient's spinal fluid injected into buttocks, and 1 cc. of colossal in spinal canal. Repeated daily. Patient died of pneumococcic meningitis.

Case 3.—Male, age 17. O. M. P. C., bilateral, from infancy. Frequent attacks of pain in left and occasionally vertigo. Two weeks ago severe attack of vertigo while working in the hot sun. Unable to stand. In bed six days with marked vertigo, left sided headache and nausea. Pain in and around the ear with mastoid tenderness. Examination showed a large subperiosteal swelling with redness and marked tenderness. No nystagmus. Large posterior perforation in drum. Foul discharge. Ear totally deaf with noise apparatus. No reaction to caloric. Ten turns to the right produced a nystagmus to the left lasting five seconds. Ten turns to the left produced a nystagmus to the right lasting twenty-two seconds. Fistula test negative. Radical mastoidectomy. Large perisinus abscess below the knee. Vestibule opened from behind and free bleeding resulted. Promontory removed between round and oval window. Highest temperature, 100. No headache,

nausea or vertigo. Released from hospital on the fifteenth day. Convalescence uneventful.

COMMENT.

Case 1 was the first patient in the writer's experience to suffer a complete destruction of the labyrinth while under his observation. Fortunately she returned for treatment before her serous meningitis became a diffuse suppurative one.

Case 2 was considered a poor risk from the start, due to his severe acute nephritis and secondary anemia. A serous labyrinthitis was diagnosed because of the slow loss of hearing and the absence of nausea, vomiting and past pointing. The caloric test could not be used because of a swollen blocked canal. On the twelfth day the ear was completely deaf, and the writer assumed that the labyrinth was also functionless. Considerable time was consumed in the second operation, and a transfusion was done on the table. The additional time required to drain the labyrinth was too much for the patient to withstand, but had this been otherwise it is quite likely that the patient's life could have been saved.

Ruttin⁸ is injecting colloidal gold into the spinal fluid in suppurative meningitis and cites three recent recoveries, two with a positive culture. Rooks⁹ reports two recoveries following the injection of autocerebrospinal fluid, both of which gave a positive culture.

Case 3, a diffuse latent labyrinthitis, had the labyrinth drained along with the radical mastoidectomy, and made an uneventful recovery.

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XXVIII.

REDUCING THE STRENGTH OF COCAIN SOLUTIONS IN NOSE AND THROAT PRACTICE.*

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The elimination of cocain as a local anesthetic from nose and throat work is still a problem. There are many substitutes on the market, but so far not one of them has proved to be of the same value as cocain, with the exception of novocain, which has replaced the cocain in cases in which infiltration anesthetic is used. However, no substitute has become popular enough to replace the cocain as a topical anesthetic. A survey of the leading institutions of New York City shows that almost all the hospitals and clinics where nose and throat work is being done use 5 or 10 per cent cocain, in some cases even 20 per cent cocain or flakes, not only for operative procedures but also for examinations. The cocain accidents, although they are reduced by avoiding the injection of cocain, still result from the use of the drug in high concentrations. What are the possibilities of eliminating or even reducing these dangers? Cocain infiltration, which prevailed until probably ten years ago, has almost disappeared; but the use of 5, 10 or 20 per cent solution, or flakes, is still very common among the nose and throat specialists. There are some who, for local tonsillectomies, still use the 10 per cent cocain swab as preliminary, and many use the same percentage in nasal and laryngeal operations.

Experience shows that local tonsillectomies can be done satisfactorily with the use of novocain-adrenalin, even in the hands of beginners. There are cases in which the cough, gagging and nervousness of the patient test the skill and the patience of the operator, and make the local tonsillectomy not an easy operation.

*From the Nose and Throat Department of the New York Post-Graduate Hospital and Medical School.

However, I am convinced that these difficulties can be overcome if the surgeon has gained the confidence of the patient and uses a good local anesthesia. The use of novocain, $\frac{1}{2}$ to 1 per cent solution, with adrenalin combined, in some cases, with bromide, a barbital preparation, or even with morphin as a preliminary, gives entirely satisfactory results.

There is no excuse for using cocain, which increases the danger to the patient's life. However, there are cases which require a topical application of an anesthetic, and in which novocain cannot be used; here we are confronted with the question of using cocain.

Braun,¹ a pioneer in the use of the local anesthetic, formulated the following requirements for a substitute for cocain:

1. The drug used as a substitute must be less toxic and have more or the same anesthetic effect.
2. The drug must have no irritating effect on the tissues.
3. It should be soluble in water, and constant in solution for sterilizing purposes.
4. It should be miscible with adrenalin.
5. For topical application, it should be absorbed by the mucous membrane.

Of all the substitutes for cocain, novocain complies with the first four requirements. As to the fifth, experience shows that not even a 30 per cent solution of novocain has a satisfactory anesthetic effect in the larynx when applied topically.

As there are no means of completely eliminating cocain at the present time, would it be possible to reduce the percentage used and still obtain the same result?

Gehse² shows that the addition of phenol increases materially the anesthetic properties of cocain and that with it the solution is much more stable. His idea is not a new one.

Bonain,³ in 1898, recommended a prescription in which he used phenol, menthol, adrenalin and cocain in equal parts, which he used mainly for paracentesis. Braun, among the Germans, and Freudenthal,⁴ in America, condemn this mixture as containing

too much phenol and doing too much damage to the epithelium. Independently of Bonain, Caesar Hirsch⁵, in an attempt to increase the stability of the cocain, added phenol, to prevent the formation of the small white fungi which often grow in cocain solutions if allowed to stand for any length of time; and to increase the effectiveness of the cocain, he added potassium sulphate. His original prescription was the following:

Cocain.....	3.0
Potassium sulphate, 2 per cent.....	20.0
Adrenalin (1.1000).....	10.0
Carbolic acid sol. $\frac{1}{2}$ per cent to make.....	100.0

There are many clinics in Europe, among them Denker's, which approved of the above solution, although some of them (Abraham) criticise the amount of adrenalin and reduce it. Hoffman and Kochman,⁷ in the Institute of Pharmacy, Greifswald, proved that the addition of potassium sulphate would not affect the strength of the cocain if it is applied topically, but would increase the effect of the novocain when it is used for injection.

Gehse, in his experiments, tested the minimum concentration of novocain and cocain, used topically, which has an anesthetic effect on the cornea of the guinea pig, and then both novocain and cocain in combination with potassium sulphate, and, as is shown below, the potassium sulphate has no synergistic effect and does not increase the potency of either the novocain or the cocain.

Concen- tration	Cocain Duration of a.	Novocain Duration of a.	Cocain Concent.	Pot. Sulph. Duration	Novocain Concent.	Pot. Sulph. Duration
2%	46 min.	20 min.	2% + 1%	51 min.	2% + 1%	28 min.
1%	20 min.	5 min.	1% + 1%	22 min.	1% + 1%	6 min.
0.5%	6 min.	0.5% + 1%	7 min.	0.5% + 1%
0.25%	0.25% + 1%	0.25% + 1%

He then tested 0.50 per cent and 0.35 per cent solutions of phenol, and proved that no anesthesia was produced by these phenol solutions; only the reflexes were retarded and slight hyperemia occurred, but no harm was done to the corneal epithelium. Then a combination of phenol with cocain and novocain was tried, and, as the chart below shows, the efficiency of the cocain was increased five or ten times, so that the 2 per cent solu-

tion paralleled an ordinary 10 per cent solution in efficiency. Furthermore, the anesthetic effect is much more rapid.

COCAIN		WITH PHENOL 0.5 %
Concentration	Duration	Duration
2%	28 min.	69 min.
1%	20 "	31 "
0.5%	6 "	33 "
0.25%	18 "
0.125%	13 "
0.0625%	7 "
0.0312%

His standard solution is the following:

Cocain.....	2.0
Phenol.....	0.35
Normal saline to make.....	100.0

Add adrenalin as required in the individual case.

There is no explanation for the synergistic effect of the phenol in combination with cocain at the present time. K. Meyer showed that leaving out the potassium sulphate from the Hirsch solution reduced the toxicity to a minimum. The mixing of phenol with other drugs, like psicain or tutocain, did not give the same result as with the cocain.

For the past four years Hirsch has been using the tutocain, phenol and potassium sulphate with good results, and has eliminated the use of cocain from his clinic entirely.

For a few months we have been using the cocain-phenol combination by Gehse in the operative clinic of the Nose and Throat Department of the Post-Graduate Hospital exclusively, and the results obtained equal those obtained with the use of 5 or 10 per cent cocain previously used.

SUMMARY.

The advantages of this combination are:

1. Reduction of toxicity with smaller amount of cocain used.
2. Stability and sterilizability of the solution.
3. Disinfectant effect of the phenol, helping to sterilize the operative area.
4. Reduction of amount of cocain, hence reduction of cost.

The odor of the phenol, which disappears a few minutes after the packing is put into the nose, is the only disadvantage.

As to the postoperative reaction, there is no harmful effect; the mucous membrane does not show any more reaction, and the healing process is as smooth and rapid as when the plain cocain is used.

124 EAST 84TH STREET.

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XXIX.

TREATMENT OF CHRONIC MAXILLARY SINUSITIS
BY ZINC IONIZATION AND IODINE BORIC
POWDER INSUFFLATION.

DR. F. L. REESE, M. D.,

PHOENIX, ARIZONA.

Most rhinologists are familiar with the method used for the ionization of zinc in the treatment of chronic otitis media.

It is not my purpose to introduce here any discussion of the principles underlying zinc ionization or of the action of free iodine on pathologic tissue.

Making a brief reference to ionization in middle ear infection, I am forced to acknowledge that I have become disillusioned as regards the curative action of this method of treatment. Feeling sure that the failure in ionization was due to the fact that the zinc solution was not brought into contact with the diseased tissue, every available device was used to insure such favorable contact. The ear canal was kept full of solution as long as thirty minutes before starting ionization. The pus was thoroughly evacuated from the middle ear either by direct suction or through minute cannulae inserted through the perforation in the drum. The air displacement method as practiced by Pröetz in his sinus filling technic, was used. In spite of all this, but ten percent of these chronic otitis cases responded to treatment. This reference to otitis media is made because I am convinced that the failure to effect cures in these cases is due to the inability of the operator to bring the zinc solution in proper contact with the diseased tissue. This cause of failure is not applicable to the treatment of chronic maxillary sinusitis. To bring the zinc solution into proper contact with the diseased membranes of the sinuses is simplicity itself, and it was due to this fact that I early conceived the idea that zinc ionization would prove a most valuable method in the treatment of these conditions. I begin boldly by saying that from my experience, I am firmly

convinced that, with the exception of carcinoma, osteomyelitis, or syphilis, all chronic inflammatory conditions of the antra are curable by this method of treatment. I do not mean that they will be improved, but that ultimate complete recovery should be the result. In my early experience, I used zinc ionization alone in the treatment of purulent maxillary sinusitis with good results. However, in the last six months, with this ionization, I have combined the use of the boric iodine powder. The results with the combined method have been more nearly complete and brought about in much less time.

The technic of antrum ionization is comparatively simple. Many of these cases come with a window in the inferior meatus either as the result of a simple intranasal or radical antrum operation. In these cases, the field is already prepared. If a virgin operative field is presented the regulation window is made into the antrum under the inferior turbinate. It is my custom to wait a few days until the operative reaction subsides before beginning ionization.

The current is supplied by the ordinary galvanic battery—the same battery that is used for ionization of the middle ear. In addition to the battery, two instruments are required; first, the antrum electrode; second, a fine silver canula with a Luer connection. As yet, there is no such electrode on the market. My electrodes are made by soldering a zinc tip on one end of an old Shelltex spectacle temple which has a metal core. A connection for the battery cord is at the other end. This is, of course, curved to facilitate its passage into the antrum.

The patient is seated in the treatment chair. A eustachian catheter is now introduced into the antrum through the window and, by air pressure, all the accumulated pus is blown out into the nasal cavity. The catheter is removed and the patient is ordered to blow his nose. Through the antrum window is now inserted the specially devised electrode. Alongside of this electrode is inserted, also into the antrum, the fine silver canula. The patient holds these in position with the thumb and forefinger, grasping the nose in such a manner as to leave one nostril open. Retaining these in position, the patient lies on a table or couch on his side with the diseased antrum down. The positive pole

of the battery is now connected to the electrode while the negative pole is attached to the leg or other part of the body. The patient is instructed to breathe easily through his mouth. With a Luer syringe, the antrum is now filled through the silver canula with the zinc solution. The current is turned on gradually, the strength varying with the individual. The average patient tolerates eight to ten milliamperes without discomfort. The duration of the treatment is from five to fifteen minutes, depending upon the reaction; using a shorter period for the initial ionization. In a few cases I have continued the treatment for as long as thirty minutes. The electrode and the canula are now removed, the patient is returned to the treatment chair, the antrum is dried by air and is now ready for the iodine powder insufflation. This may be done immediately following the ionization, although I am not inclined to use the powder until the day following. This is done with an ordinary powder blower through a eustachian catheter. About ten grains are used in a treatment.

CASE REPORTS.

Mrs. T., aged 37, who had a bilateral maxillary sinusitis, had been operated upon three years previously. If permanent windows had been made, they were closed, as I had to make new openings into the antra. This patient suffered from a constant pus discharge from both nostrils. She was first seen September 1, 1929. Ionization was employed every third day until October 5th. Treatment stopped for two weeks on account of intervening cold. When seen again in November there was a slight discharge; no odor. The antra was ionized every third day for four consecutive treatments and the patient discharged as cured.

Mrs. O., had a bilateral sinusitis of many years' duration. She gave a history of chronic discharge from both nostrils. Polypi had been removed from her nose as often as every day; had been under the care of one of the best otolaryngologists for the last two years. She was receiving constant treatment. In spite of the fact her nose had been completely cleaned of all polypi both antra as well as the ethmoid region were full. When first seen by the author ionization was used every third day for six weeks, at which time she was dismissed as cured. She returned to my office six months later for inspection of her nose. At this time no pathology was found.

In conclusion, I would say that I consider that all cases of chronic maxillary sinusitis, with the exception of those conditions mentioned at the beginning of this article, are curable with the combined ionization and iodine boric powder insufflation.

XXX.

TAKING STOCK OF THE RADICAL MASTOID OPERATION.

HARRINGTON B. GRAHAM, M. D.,

SAN FRANCISCO.

On the whole the radical mastoid operation has stood the test of time far better than a great many other operative procedures and there have been but few improvements in the technic of thirty years ago. In general, it is a pleasure to see one's cases come back for inspection in spite of the fact that sometimes the patient complains about having to have assistance with a periodical house cleaning. The danger to life has passed and the surgeon can truthfully say that over the long period of time he has stopped the degenerative process which would account for a deafness progressive in character.

Without going into minute detail, I wish to review a few of the salient points connected with the operation which may be of interest.

It has been said that a suppuration which has continued for more than six months in a child is a good reason for considering a radical mastoid operation. If the word mastoid were left out of this picture, I should say that the statement was nearly correct.

In my early experience I had occasion to remove the adenoids and tonsils of some fifty children under the age of 14 who had been suffering for an indefinite number of months or years, with suppuration of the ears. In only one instance in these cases did I fail to obtain a cessation of the drainage, and in this case I made a diagnosis of tuberculosis, later did a radical mastoid operation and procured a satisfactory result.

Children are a law unto themselves and will respond very quickly if given a little help. An irritation, such as a nasal or throat infection or a constitutional trouble such as lues or tuberculosis, is readily communicated to the ear; and the ear trouble disappears readily under treatment of the mother trouble. So

thorough study of the individual is an essential, but often neglected, duty of the surgeon.

Infants of a few weeks often show early signs of ear infections which, if attended to at once, will save the hearing of the patient. The indigestions, rises of temperature, and restlessness seen in pneumonia and the cervical adenitis of early tuberculosis are only a few of the signs that should lead to early examination.

In my experience, the age of an infant, however, is no bar to a radical operation if once it has been determined on, as in tuberculosis. Instance the case of a six months' old infant with a pure culture of *b. tuberculosis* in the pus from the ear and cervical adenitis. At operation the middle ear was found filled with granulations and there was a necrosis of the whole mastoid bone. A thorough exenteration was made, the glands of the neck were removed at the same sitting. The patient made an uneventful recovery and at present is the mother of a couple of healthy children. The hearing in the operated ear is good though, of course, not perfect.

If in this type of case the bone is removed to the dura in all directions, the chances of recovery are enhanced and the possibility of a tuberculous meningitis, induced through the exposure, is remote.

Infants react very quickly to the introduction of the tubercle bacillus, bone especially breaking down readily, and the mastoid is no exception to the rule; if, therefore, the trouble is not recognized very early and a radical exenteration done, even though the drum and small bones are saved temporarily, disaster will result. The one redeeming factor in the invasion here is that as a rule it is of the bovine type and, therefore, the least virulent.

The possibility of lues must never be overlooked in infants, and when there is any doubt, a course of antiluetic treatment will do no harm and may save a lot of work. It is surprising how quickly an operated patient will recover following mixed treatment after a long siege of delayed local treatment. Lues of the middle ear in infants does not present the fulminating destructive lesions that tuberculosis does and the possibility of a tolerably good function after treatment is much better.

If there is no lower limit to the age when a radical operation may be undertaken, I think there is also no upper limit, other things being equal. With the present day method of local anesthetic for the radical operation so perfectly developed, one need not hesitate on account of shock. I have never been reproached by the patient after the ordeal and, in one instance, an elderly lady came back for an operation on the other ear as her comfort had been increased so much by the former one. Patients of 80, who otherwise are in good health, seem to stand the radical operation with little ill effect. Healing in these elderly patients does not seem to be delayed and is as complete as in younger people. In most instances hearing is improved immediately and permanently, for the labyrinthine degeneration has seemingly reached its limit and one is dealing with a conductive interference.

The selection of the anesthetic depends entirely on the mental and physical condition of the patient. A local anesthetic may be given to a very young individual who is extremely low if the operator is rapid in his work and, on the other hand, gas may be administered in old age if the mental attitude of the patient is such as to preclude a local anesthetic. In all local cases, I think it is better to give gas for the plastic work and for clearing out the middle ear, though this is not necessary. In administering the local anesthetic, after injecting the skin and subcutaneous tissues, the needle is carried beneath the periosteum and a liberal supply of novocain injected, special attention being paid to the external auditory canal.

One of the greatest hindrances to an early termination of the case is an insufficient lowering of the external portion of the facial ridge causing a pocket to be formed at the former apex for the accumulation of wax and debris under which an ulcerative process continues for years. Better remove the entire apex and allow the muscle to partially fill the cavity than leave a recess which cannot be easily reached from the external canal. A wide plastic opening, though somewhat unsightly, will aid in those cases when the facial ridge is not lowered or the apex is shallow.

The advisability of leaving the drum and small bones when the hearing is good and the lesion apparently in the upper por-

tion of the bone, is a question that must be decided by the social status of the patient and the pathology present. The patient has a right to expect every improvement possible in hearing consistent with a healed wound and eradication of the danger of an extension of the process to the intracranial contents with its threat to life, but one could take more of a chance with a patient who will remain under his observation indefinitely, and who is dependent upon a keen hearing for his living, than he would with a laborer of small mentality who was expecting to live at a great distance from medical care. In the one case a modified radical operation could be tried, even when the chances of success were meager, whereas in the other case it would be folly to attempt it even though the probabilities were on the side of success.

That a modified radical operation of the Bondy type is fully justified, even in the presence of a cholesteatoma, has been amply demonstrated, as instance a case when the diseased ear was the poorer hearing ear and ten years following the operation had remained not only perfectly dry but was considered by the patient to be his more serviceable ear. It is just as fallacious to condemn this operation as it is to try it in all cases when the small bones and drum are visible.

For some unknown reason, perhaps a constitutional dyscrasia, some ears continue to secrete for months and even years following the operation. Many of these need only patience and care on the part of the operator, not a reoperation; eventually they will become quiescent. Of recent years the boric iodine powder has considerably lightened our troubles, wounds healing under its use which have annoyed us beyond measure.

In my experience, the presence of lues and tuberculosis in adults as factors, hindering the convalescence has been much neglected. Lues, especially, is easily diagnosed with our present day methods, and a course of preoperative treatment is essential to a successful outcome of the operation.

The after treatment in tuberculosis is markedly influenced by the routine treatment for tuberculosis of the lungs, showing that the local lesion is only an evidence of the general disease.

Local tuberculosis of the middle ear is, as I have formerly pointed out in a paper before the Triological Society, much more

frequent than we generally acknowledge; it is simply hard to demonstrate and most of us do not make the routine examinations to uncover its presence. Bacteriologic examinations of the pus, guinea pig inoculations, examination of the tissue removed, and observation of the patient, take time and labor for which the patient has often not the money nor the operator the time. The treatment of the local process is the same, save that with the knowledge of the presence of local tuberculosis the operation would be more extensive, leading to a removal of the tip and an uncovering of the dura.

There are so many factors entering into a discussion of the effects on hearing of the radical mastoid operation that it is rather confusing, but fundamentally the better the hearing before the operation the better it will ultimately be following the operation; this statement carries with it a comparative estimation of the receptive power of the labyrinth. In very young people with a good labyrinth and a marked interference with the conductive apparatus, there should be an improvement in hearing, whereas in case the middle ear is not markedly involved in the process, the hearing should be considerably decreased.

Immediately succeeding the operation, there is often a cushion of granulation in front of the promontory wall which interferes with hearing but which disappears in six months' time leaving a thin epidermal covering and remarkably good hearing. The disappointment of the patients in these cases, after one has discharged them as dry, often leads to much dissatisfaction which can only be allayed by time. After a skin graft this does not take place.

In adults, generally speaking, the hearing one year following the operation is decreased, but many an operation has been done to increase the hearing or, at least, to save what remnant there was left. The modified radical operation is certainly indicated for the conservation of hearing when it is possible to demonstrate a continuity in the bone chain and is worth the extra effort.

The enthusiasm with which the labyrinth was formerly opened has somewhat subsided. That every dead labyrinth must be opened when a radical operation is done in order to prevent an acute exacerbation of a labyrinthitis with possible meningitis

and death is to be questioned. The danger to the facial nerve with resultant deformity to the patient is so great in this operation that one hesitates to undertake it unless there is a well marked indication. Drainage may be obtained by a simple removal of the promontory and a suppurating labyrinth is not so terrifying as a crooked face for the remainder of one's life.

A labyrinthitis at the time of a radical operation, with nausea, nystagmus, deafness, all signs of a suppurative process, has in two instances with me become a well functioning labyrinth in years following, showing a recuperative possibility that must be taken into consideration, and to say that these were only serous labyrinthitis because they recovered is begging the question.

Since the presentation of this paper there has appeared in the December ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY an article by Dr. Lewis Fisher, describing a case of suppurative labyrinthitis which, apparently, showed reaction before death and in a personal communication he says: "I could not, however, very well assume such a collection of pus to be a cerebellar abscess complicating a suppurating and purulent labyrinth, for the reasons that you suggested, namely, the presence of some function within that ear. The autopsy, of course, proved that he did have suppurative labyrinthitis, and I believe that we will have to revise our idea that a suppurative labyrinth must in each and every instance destroy the function absolutely and completely."

Since Dr. Sterling Bunnell has demonstrated that it is possible to successfully repair a damaged seventh nerve even years after the injury, more attempts will be made, even in desperate cases, to relieve a tragic misfortune. His last personal report is of a transplant from the sural into the facial nerve. The outcome of this will be watched with a good deal of interest. In a patient sent to me with intratympanic damage I successfully repaired the nerve by uncovering it for its full length and placing the fibres as close together as possible. There were evidently uncut fibres at the lower portion of the nerve which assisted in keeping the ends in place, although the paralysis had been complete for three months. One year after the operation there was nearly normal movement of the whole face. It is quite possible that many of these paralyses are not due to a severance of the nerve

but to pressure of a depressed fracture of the canal, and a freeing of the nerve from the canal may be enough to repair the damage. This is one of the most difficult branches of surgery, requiring infinite care and much practice with small needles on nerves of animals and, according to Dr. Bunnell, should not be undertaken without the requisite training. If the nerve is freed along the whole fallopian canal, a great deal of slack may be obtained and ends be brought together which appear impossible to unite.

It seems to me that the radical mastoid operation is becoming less frequent, not because it is less popular but because it is less frequently indicated. The early attention to defects of the nose and throat, early paracenteses in pneumonia and exanthematous diseases, and disturbances of digestion in infancy, are the leading factors in the prevention of suppurative processes in the ears. Many operations in later life are due to neglect and could certainly have been prevented by early medical care. I believe that the lessening of tuberculosis has helped immensely in decreasing the incidence of chronic suppurative otitis. Extra care in the exanthemata and pneumonia is still further going to help and the early attention to the ears of nursing infants will make the radical mastoid operation a rarity.

490 POST STREET.

AN UNUSUAL SERIES OF LATERAL SINUS CASES.

S. B. CHASE, M. D.,

FORT DODGE, IOWA.

This series of cases is of interest mainly because of the high incidence of the complication of infective lateral sinus phlebitis or sinus phlebitis with thrombosis in the series. It was the only primary complication present. It tends to show the selective action of certain strains of streptococci for certain body tissues.

In this series of cases, six of the cases were operated upon by me. The diagnosis was proven correct in all cases, by section of the sinus wall, or the wall and thrombus, by a competent pathologist; in one case the clinical course and findings were fairly conclusive; in one case in which the preliminary mastoidectomy was done, but in which, for financial reasons, the patient was transferred, the diagnosis was confirmed at operation by a pathologist when the sinus was operated upon by another surgeon.

Because of the fact that an unusually high incidence brings in an element of doubt in the tentative diagnosis of a condition it might be well, in briefly reviewing this series of cases, to bring out a few points in diagnosis that seemed to us to be of value. Any patient with an acute suppurative otitis media and mastoiditis, that either before or after operation, while pursuing a fairly average course, suddenly has a rigor followed immediately by an abrupt and marked rise in temperature, with a positive blood culture, tenderness along the anterior border of the sterno-mastoid, and a metastatic inflammation, say in the ankle joint a short time after, presents no difficulty in diagnosis; but unfortunately, we do not always have such a clear-cut picture. There may be a recent valvular heart condition; pus in the urine; no metastasis may be discernible; the blood culture may be negative; there may be no obliterating thrombus in the sinus which would give a positive Queckenstedt; the temperature may be high and continuous. We hesitate to block the return flow from one side

of a patient's head permanently, unless we are reasonably certain of our diagnosis.

In this series two symptoms were marked in all cases: (1) Tenderness over the cervical course of the internal jugular vein, most marked in the region of the mastoid; (2) localized pain and tenderness over the course of the sinus through its bony groove. A thorough and experienced internist or pediatrician ruled out all other sources of the infection. The Queckenstedt test was not used in any of this series, because in the bilateral cases the symptoms definitely pointed to the side involved and in the unilateral cases the diagnosis was positively reached without its aid.

As to procedure, in event of this complication, it is nearly universally recognized that ligation of the internal jugular with obliteration and drainage of the infected portion of the sinus is indicated as soon as a diagnosis is made. Probably the most helpful adjunct to this is whole blood transfusion, as it immediately introduces fresh and active antibodies, and replaces with healthy blood cells and fluid that which has been weakened and destroyed by the infection.

The average recognized mortality with operation of this complication is 50 per cent, and it is around 90 per cent without operation. The mortality is much higher in cases of infective sinus phlebitis than phlebitis with thrombosis, either with or without operation. In speaking of 50 to 90 per cent mortality, I refer to all cases of this complication—that is, phlebitis and thrombophlebitis. Our series bears out the preceding. In sixteen previous cases our mortality with operation was only two cases which was much too low, but the cases were scattered over a period of several years, and nearly all were cases of marked thrombophlebitis in which, in my estimation, and I believe in the experience of others, the mortality is much lower. Therefore, a more or less obliterating thrombophlebitis seems to indicate a less virulent organism with a strongly reacting individual, which naturally, when aided by operation, should show a higher percentage of recoveries.

The incidence of sinus involvement in our series of 29 cases was nearly 30 per cent, seven of which were bilateral, or 36 mastoid infections. It is this unusually high incidence of the compli-

cation that makes this series of interest. This series ran over a period of seven months, from January 1, 1930, to August 1, 1930. The cases were not from any one locality, but from territory averaging about 100 miles in width.

A brief résumé of the cases in question follows:

Case 1.—Howard P. Past history negative except was struck on head in auto accident two months previous to admission. Cleared up entirely following accident. Family history negative, except one brother had died twelve months previously, aged nine months, of pneumonia.

Present history: December 26th, patient had severe cold with fever, followed by earache. Drum ruptured on the 28th and pain became less. Discharge profuse. Temperature continued. January 1, patient was referred by pediatrician.

P. X.: Well-nourished lad, about $4\frac{1}{2}$ years of age, who appears very ill. Temperature 103.5, pulse 140, respiration 24. There is a profuse serosanguineous discharge from the right ear. Mastoid presents marked bony tenderness. Enlarged cervical glands, bilateral. Posterior superior canal wall sagging. Marginal tympanic perforation in posterior superior quadrant. Balance of examination negative. Urine: Trace of albumen and 2 plus acetone. Blood count, 17,000; 80 per cent polys.

Diagnosis: Acute suppurative otitis media, mastoiditis, right.

X-ray: Cellular type of mastoid, right, all cells blurred; some destruction of bone. Culture from canal: Hemolytic streptococci. Treatment: Right mastoidectomy, gas anesthesia; all cells obliterated; no dura exposed; sinus not exposed. Mastoid closed; tube drain inserted. Culture at operation same as from ear canal.

Postoperative course: January 2nd, high temperature, 105.8, pulse 140, respiration 38; low temperature 100, respiration 22, pulse 110. This approximate curve continued to January 4th. Wound in good condition; no erysipelas or cellulitis. January 4th, septic infarct appeared at edge of left pinna, about 2 cm., at outer edge; also metastatic abscess in scalp in right occipital region (area of deep cellulitis), circumscribed, about 4 cm. in diameter. Later both sloughed, leaving a clean-cut V-shaped notch in edge of left pinna, as though cut out with a knife, and a deep punched out ulcer in right occipital region. Blood culture nega-

tive. Marked headache in the right parietal region and tenderness along anterior border of right sternomastoid. No chills. The general examination is negative. The X-ray of the chest was negative; throat negative. Eye grounds negative. No cervical rigidity. No vertigo or vomiting. Child bright, but restless. Blood count 10,600, polys 86, some new white cells, reds 3,250,000. Kahn test negative.

Diagnosis: Infective lateral sinus phlebitis; general septicemia.

Operation: Ligation of right internal jugular, drainage and obliteration of the sigmoid sinus. At operation the sinus wall appeared thickened and dark red. Bony cover of sinus appeared normal. No thrombus present. Inner wall of sinus appeared dirty gray in ascending portion. Free return flow of blood from distal and proximal ends. Patient stood operation well. Very little shock. Pathologist's report: Infective sinus phlebitis. January 5th, temperature 100 to 106, pulse 120 to 140, respiration 20 to 30. Whole blood transfusion given. January 6th, temperature 99 to 104, pulse 110 to 120, respiration 20 to 28. General condition fair. Urine negative except slight trace of albumen and acetone, which continued throughout illness. Mastoid and neck in good condition. No cellulitis. Canal dry. General examination negative, except patient pale, showing marked secondary anemia.

Summary: Patient ran irregular septic course, low grade at times, with transfusion of whole blood on January 13. On January 23rd, neck reopened and jugular ligated as near subclavian as possible, in hope of getting below the area of infection. Septic course running higher. Temperature reached 105 daily; no new metastasis found. Patient died February 1st; apparently terminal meningitis and asthenia. Postmortem: Brain negative except purulent exudate at base in region of bulb.

Case 2.—Edna R., white, female, age 16, schoolgirl, American. Past family and social history, nothing of interest bearing on the case. Present history: Patient had measles ten days before admission; fairly severe. Some fullness in ear and deafness. Two days before admission patient complains of increasing deafness. Pain in ears which became severe. Patient admitted February 16, 1930.

P. X.: General, negative, except some reddened areas on skin from measles. Ears: Both tympanic membranes red and bulging. Sagging in posterior superior wall. Slight tenderness at tip of mastoids. Temperature 101.4, pulse 110, respiration 24. Blood count 14,600, 80 per cent polys. Urine: Slight trace of albumen and acetone. X-ray of mastoids, cellular adult type; slight blurring; no destruction of bone. Diagnosis: Acute suppurative otitis media. Mastoiditis (?) Treatment: Bilateral paracentesis, gas anesthesia. Cold to mastoids for 48 hours. Pus present in both middle ears on paracentesis. Culture: Pneumococci. Because of the increasing pain and tenderness over mastoids, in spite of free drainage and increasing pathology, as shown by X-ray, both mastoids were operated on February 20th. Usual operation. No complication at operation. Temperature dropped to approximately normal in 48 hours. Patient discharged to dressings by home physician on February 28th. Patient had fairly normal postoperative course at home and was seen at weekly intervals by myself. Both mastoids healed well, canals became dry. On March 25th, patient began to have some pain on left side of head, not severe and left canal again became moist. Slight temperature not to exceed 100.5. This continued until April 7th, when she was again brought to the hospital.

Examination: Right mastoid completely healed. Left, small wick drain in mastoid; considerable pus in ear canal. Patient complained of severe pain in left mastoid region extending back towards occiput. Marked tenderness along anterior border of left sternomastoid. Temperature 104, pulse 130, respirations 30. General examination negative; local negative, except as above. Eye grounds negative. Urine negative. Blood count 12,500, 88 per cent polys. Pain continued very severe in left parietal and suboccipital region. Tenderness persistent in left neck. No aphasia, no chills, blood culture negative, spinal fluid negative.

April 11th. Gas anesthesia. Left internal jugular ligated at sup. thyroid. Sinus opened and extensive obliterative thrombophlebitis found. Sinus was obliterated from region of bulb; no return flow from proximal end or any collateral. Incision carried backward and sinus found thrombosed nearly to the torcula. Pathologist's report: Infarct of brain and embolism.

Subsequent course: Patient threw septic embolus into right knee joint and base of right lung. Blood culture positive; streptococci. Both formed abscesses. The first was drained; the second ruptured into the pleural cavity, formed an empyema; was drained by rib resection. After a long, stormy, septic course marked by profound anemia and accompanied by repeated transfusions of whole blood, patient left hospital on May 24th, after ten days of nearly normal temperature, with mastoid nearly completely healed, knee completely healed and slight drainage from right lower chest.

Case 3.—Viola B., age 14, female, white, schoolgirl, American. Past medical, family, social history, negative. P. I.: Patient became sick a week before admission with symptoms of acute coryza. High temperature, chills, copious nasal discharge, sore throat, pain in right cheek, frontal region, right ear, mastoid. Two days before admission patient became stuporous. Could be roused with difficulty. No vomiting or paralysis. Profuse purulent discharge from left ear and nares. Admitted to hospital, March 24, 1930.

P. X.: Patient, white, female of about stated age. In stuporous condition. Can be roused with difficulty. No cervical rigidity, Babinsky, etc., no paralysis. Pupils normal, round, equal and react to light, fundus negative, except area of reddening and tenderness in left ankle. Profuse purulent discharge from right nose and ear canal. Sagging of posterior superior wall, right, and central perforation of drum. Slight edema at tip of mastoid, right. Left ear apparently normal. Throat negative except much pus in nasopharynx. Temperature on admission 105.8, pulse 135, respiration 34. Spinal fluid showed much globulin, 95 cells, 90 per cent polys, Kahn negative. Blood count, 17,700; urine, few granular casts. X-ray sinus: All blurred, right. Mastoid X-ray, right, cellular type, all cells blurred, some bone destruction. Culture from ear and nose showed strep. Blood culture taken, reported in 48 hours, negative.

Diagnosis: Acute suppurative otitis media and mastoiditis, right. Acute suppurative pansinusitis, right, toxic encephalitis.

Treatment: Right mastoidectomy, drainage of all sinuses, right. Patient stood operation well.

Subsequent course: Patient continued to run high septic course of fever, never getting below 101, usually reaching 105 or more daily. Stuporous condition continued, although patient would rouse up and recognize parents and friends and make known wants. No new neurologic symptoms developed. On April 1st, blood culture was positive, strep. Ligation of right internal jugular done and drainage and obliteration of sinus, right. Sinus wall dull and gray, considerably thickened. No granulations, pus or adhesions to bony wall. Inner wall of sinus showed thin grayish flocculent exudate. No thrombus present. Free return of blood from collaterals and both ends. Section of vein showed "infective sinus phlebitis." Course continued the same. On April 6th, blood culture still showed hemolytic strep, in tremendous numbers. Spinal fluid on March 27, 1930, showed plus globulin and 35 cells, 90 per cent polys, and on April 6, 1930, showed increased pressure, slight trace globulin, 3 cells. Red cells would get down to around 3,000,000 with low hemoglobin. Whole blood transfusions on March 28, 1930, April 3, 1930, April 6, 1930. Patient's temperature rose to 107 on April 10, 1930. Patient expired. Postmortem: Gross of the brain, no abscess, redness and congestion over all pial membranes. Entire brain congested. No pus. Microscopic examination not done.

Case 4.—Mrs. Carrie L., age 37, married, white, American, multipara. Family, social, personal and past history not important, except patient is overworked, undernourished.

P. I.: Following severe cold and sore throat, patient developed pain and tenderness in right ear, deafness, followed in 24 hours by profuse serosanguineous discharge from the right ear canal. Severe pain in right mastoid region with marked tenderness. No vertigo, chills, nausea. Patient admitted to hospital March 29, 1930.

P. X.: Undernourished white female, about stated age. Apparently in severe pain. General examination negative. Eyes, nose and throat and left ear negative. Right ear shows profuse purulent discharge running from canal down over face. Extreme mastoid tenderness and some subperiosteal edema. Posterior canal wall sagging and pus pulsating through perforation in post. quad. of drum. Temperature 100, pulse 88, respiration 22.

Blood count 19,400, polys 84 per cent. X-ray of mastoid, cellular type, all cells blurred with destruction of bone. Culture from canal: streptococcus. Kahn negative.

Diagnosis: Acute suppurative otitis media and mastoiditis, right.

Treatment: Simple mastoidectomy, right. Gas anesthesia. Usual operation and nothing of note at operation. Sinus and dura not exposed.

Postoperative course: Patient went along with about the usual postoperative course. Up out of bed on the third day; complained of restlessness and some pain in right head. Temperature was approximately 100.2 on third day; on the sixth day temperature reached 102. General and local condition without much change. On April 6, 1930, patient complains of some pain in right neck. Pain on swallowing and headache in right parietal and suboccipital region. Examination of throat negative, but marked tenderness along anterior border of right sternomastoid. Later in the day patient had a rather severe chill and temperature rose to 103. A few hours after chill, patient began to cry and groan with excruciating pain in lower limbs. Course of pain followed the entire sensory tract of both lower extremities, from hips to toes; worse left. Pain was severe and constant and could not be controlled by opiates. Temperature dropped, April 7, 1930, to maximum 100.4.

Diagnosis: Septic embolus in lower posterior portion of spinal cord. Lateral sinus phlebitis, right.

Because of the hopelessness of the spinal cord complication, blood culture was not taken, spinal puncture was not done because of the danger of hitting the infected infarct and the sinus was not operated upon. There were no symptoms of central infection, such as headache (except as localized along the course of the sinus), cervical rigidity, eye grounds or eye muscle changes, no central motor symptoms. Patient's mentality perfectly clear. Twenty-four hours later the pain in the lower extremities continuing constant and extreme, the patient's temperature went to 102.8 maximum, dropped to 99.8 the following day. Symptoms remained the same, and on April 10, 1930, the patient developed an ascending motor and sensory paresis which

continued upward, hour after hour, and patient died of a central cardiac and respiratory paralysis in about eight hours. The infarct had undoubtedly ruptured into the canal with a resulting suppurative involvement of the cord, the usual result of an acute cord abscess. From the course of the disease and its symptoms, I think there is no doubt that the patient had an infective lateral sinus phlebitis, and I am therefore presenting it in this series, although it is the only case that was not operated upon and the diagnosis confirmed by the pathologist.

Case 5.—For the sake of variety, I am going to give in the next case the pediatrician's history verbatim, adding a few notes of my own.

Christy W., age 6, white, female, schoolgirl. Chief complaint: Earache. Pain over the left ear. Family medical history not important. Past history not important. Present illness began about two weeks ago with severe cold and pain in left ear. This earache subsided and since that time she has had fever. Two days ago she had high temperature with chill. Since that time the earache and pain over the side of the head have increased.

Physical examination shows a well developed and nourished white female, aged six years, having a temperature of 101, pulse 134, respirations 24. There is marked tenderness over the left side of the head as well as the mastoid region, and this tenderness extends downward along the course of the jugular vein. Left ear drum red and bulging. Cervical glands enlarged, 2 plus on the left and 1 plus on the right side of the neck. Heart, lungs, abdomen and extremities negative. W. B. C. 17,000, with 76 per cent polys. Urine negative except for a trace of albumen.

Diagnosis: Otitis media acute, left. Mastoiditis, left. At 11 a. m., on May 17, 1930, mastoidectomy was done by Dr. Chase. May 18th, the patient still complains of severe pain in left ear. Temperature has been as high as 100.2. May 19th, patient vomiting. Temperature up to 103.8. Still complains of severe pain in the left ear. May 20th, blood culture was taken. Mastoid culture remains sterile. W. B. C. 16,700. May 21, highest temperature in the past 24 hours was 101.2. Patient does not look well, vomits occasionally and still complains of pain in the left ear. May

22nd, temperature suddenly rose to 104.4. Patient is still complaining of pain. Vomiting some. No new signs made out except continued tenderness over the left side of the head and the left neck. May 23rd. At 5 p. m. left jugular was ligated and left lateral sinus was blocked off by Dr. Chase, the thrombosis extending from low in the neck through the sinus beyond the region of the original mastoid operation. This thrombus was quite well organized. Blood culture was taken. May 24th. Blood culture taken yesterday was positive for hemolytic streptococci. Patient, a type 2. Was given a direct transfusion of 220 cc. of whole blood from the father. There was no febrile reaction following the transfusion. Patient did develop a profuse urticaria over the face, which was relieved by adrenalin. Highest temperature today, 101. May 28th. Since transfusion on May 24th, patient's highest temperature 100.4. Is taking food and fluids well. Mastoid wound is healing satisfactorily. June 2, 1930. Patient's temperature has remained normal. Wounds are healing satisfactorily. Patient was allowed to go home. Tissue report (by A. C. Starry, M. D., Sioux City, Iowa): "A portion of the lateral sinus wall which was presented for examination showed a marked fibroblastic proliferation and an organized thrombus." Diagnosis: Otitis media, left, acute. Mastoiditis, left. Lateral sinus thrombosis, left. Septicemia, strep.

Notes.—This was a case of so-called primary jugular bulb thrombosis. There was a history of otitis media two weeks prior to admission. No discharge and following this the patient ran an irregularly septic course. On admission there were no middle ear or mastoid symptoms present. The drum appeared normal. There was no tenderness or edema over the mastoid. X-ray of the mastoids was negative. There was nothing except tenderness over the vein in the neck, localized headache, pain over the course of the vein in the skull. Blood culture was negative on admission, positive following operation. At operation the superficial mastoid cells appeared normal, but as the sinus was reached there was a small amount of pus in its region, the cells apparently having been infected from the sinus. An obliterative thrombophlebitis was found extending from down in the neck, nearly to the torcula. According to Kopetsky, the infection of the vein may occur

(a) the internal auditory vein, (b) through the floor of the tympanic cavity, (c) the anterior wall of the tympanic cavity through the veins of the carotid plexus. The child was transfused at once, became much better immediately following, and had an uneventful recovery.

Case 6.—In this case I will again give the pediatrician's report in full with a few notes of my own.

Allen O., age 4, white, male, American. Chief complaint, earache, coughing. Family history not important. Past history not important. Present illness: Eleven days ago broke out with measles. Was quite sick for about nine days before the eruption appeared. Rash lasted about four days. Since that time the patient has been very sick and eats poorly. Highest temperature noted at home was 102. Two days ago patient complained of earache. Physical examination shows a fairly well developed and nourished white male, 4 years of age. Temperature 101.4, pulse 100, respirations 22. Right ear drum is red and bulging. There is marked tenderness over the right side of the head and along the jugular in the neck. Cervical glands enlarged 2 plus on right and 1 plus on left. Throat shows a moderately inflamed pharynx and tonsils. Heart, lungs and abdomen negative. June 4, 1930. X-ray shows mastoid involvement on the right side. Urine shows 2 plus albumin. W. B. C. 18,500 with 67 per cent polys. The earache and tenderness over the head and neck continue. At 3 p. m., right mastoidectomy was done by Dr. Chase. Patient's temperature at that time was 104. June 5. Temperature rose to 106. Patient complaining of pain over right side of head and in the right ear. Patient is irrational. Temperature remaining high, between 103 and 106. June 6th, at 4:30 p. m., ligation of right jugular and blocking off right lateral sinus was done by Dr. Chase. A phlebitis without thrombosis was noted. June 7th. Blood culture taken on June 5th was negative. Temperature this morning rose to 104. Transfusion was decided upon. At 10 a. m., patient, a type 4, was transfused from the father, a type 4; 150 cc. were given. There was a severe febrile reaction from the transfusion. Temperature reached 106.8. June 8th. Highest temperature today 104. Blood culture yesterday was negative. Patient's general condition is improved. June 9th.

Highest temperature 104. Hb. 52 per cent, R. B. C. 4,000,000, W. B. C. 11,200, with 67 per cent polyps. Another transfusion was advised but refused by the parents. June 10th. Highest temperature 103.2. Patient apparently progressing satisfactorily. Taking food and fluids fairly well. Parents refuse transfusion and want to take child home. June 11th. Patient discharged from the hospital under protest. It was learned in August, 1930, that the patient had made a satisfactory recovery. Diagnosis: Otitis media, right, acute. Mastoiditis, right, acute. Sinus phlebitis, lateral, right. Tonsils and adenoids present.

Notes.—This was a case of acute hemorrhagic mastoiditis, presenting the usual picture. The sinus was not exposed at operation. At operation on the vein and sinus, the sinus wall showed marked thickening and congestion, and again the grayish flocculent exudate on the intima but no thrombus. Section of the vein wall again "showed fibrous and acute inflammatory change." Again there was marked tenderness over the jugular and along its course in the head, easily demonstrable even in a child of four. Blood culture was negative.

Case 7.—Doris S., age 6, white, female, American. I will again quote Dr. Stahr, the pediatrician (June 7, 1930).

Chief complaint: Pain in left ear and in left elbow and shoulder. Family history not important. Past history not important. Present illness began about two weeks ago when patient had a severe cold. About ten days ago began to complain of pain in the left ear. Was seen by a physician, who diagnosed the condition as a severe cold with an ear infection. Gave patient some drops to use in the ear. Since that time patient has had intermittent earache, vomiting and fever. Today began to complain of severe pain in the left elbow and shoulder. Had a chill. Physical examination: On admission to the hospital showed fairly well developed, rather poorly nourished, very pale white female, aged six years, having a temperature of 104, pulse 160, respirations 30. Left ear drum red, thick and bulging. Marked tenderness over the whole left side of the head and neck. Cervical glands enlarged 3 plus. Tonsils are large and scarred. No postnasal discharge noted. Heart and lungs negative. Abdomen negative. Any movement of the left arm causes severe pain in the left

elbow and shoulder. There is no marked swelling, but there is definite tenderness in the soft tissues about the left elbow. Lab. Urine, albumin 1 plus, otherwise negative. Blood, Hb. 53 per cent, R. B. C. 3,530,000, W. B. C. 10,000 with 79 per cent polys. X-ray shows involvement of the left mastoid. June 10, 1930. Dr. Chase did a mastoid on the left side, tied off the jugular and blocked off the lateral sinus. The vein wall showed marked phlebitis without thrombosis. Following operation temperature remained high. Patient continued to have pain in the left ear and shoulder. June 11th. Blood culture taken on June 10th was positive for hemolytic streptococci. Patient, a group 4, was given a direct transfusion of 220 cc. of whole blood from the father, also a group 4. Patient had no transfusion reaction. Continues to have pain over the left side of head and in the left ear as well as in the left elbow and shoulder. June 13. During the past 48 hours highest temperature was 102. Condition is satisfactory. June 14th, at 12:15 a. m., the patient pulled pack out of the lateral sinus. Had a severe hemorrhage. Dr. Chase was notified and took patient to operating room, where he repacked the lateral sinus. R. B. C. down to 3,900,000. Patient was transfused at 11:45 a. m.; 250 cc. of whole blood from the father. Patient had a marked febrile reaction following the transfusion. Temperature rose to 105.6. June 16th. Patient still having some fever, up to 102. General condition is good. Taking food and fluids well. The pain in the left elbow and shoulder is decreasing. June 18th. Highest temperature yesterday 101. Patient discharged from hospital today. Diagnosis: Otitis media, acute, left. Mastoiditis, acute, left. Lateral sinus thrombosis, left. Septicemia, streptococcic hemolytic. Secondary embolic infections in the soft tissues about the left shoulder and left elbow.

Notes.—This was a case which had nearly all of the classical symptoms of sinus phlebitis when first seen. Consequently, the jugular was ligated and sinus obliterated at the time of the mastoid operation. The vein again had the characteristic appearance of phlebitis without thrombosis, and section of the vein by the pathologist proved this to be the case. She ran a long, tedious, septic course after leaving the hospital, but finally recovered. I saw her recently and she appears in good health, but had symp-

toms of some circulatory disturbance in the left temporosphenoid region, namely, a mild aphasia for reading, writing and spelling, all three of which she had been unusually apt at before her sickness. Her mother reports, however, that she is rapidly improving in this respect.

Case 8.—Clara H., age 19, white, single, German. Family, social and previous medical history not important. P. I.: Following a severe cold and pharyngitis four days before admission, accompanied by high temperature and pain in right ear, marked prostration, the right ear commenced to drain profusely and the patient complained of severe localized pain in mastoid region. Marked chill second day after onset. Admission to hospital, July 24, 1930.

P. X.: Patient white, female, of about stated age. Apparently in severe pain and greatly prostrated. General examination negative. Local examination: Profuse discharge from right ear with tenderness and edema over mastoid. Sagging in posterior wall and perforation in posterior quadrant of drum; considerable soreness and cellulitis in right. Eyes, ear, left, nose and throat otherwise negative. Culture from ear showed streptococci. Blood count 15,000 with 80 per cent polyps. Urine: Much albumin, acetone trace, numerous red blood cells, no casts (not cauterized).

Diagnosis: Acute suppurative otitis media and mastoiditis, right.

Treatment: Simple mastoidectomy; no complications. Post-operative course: Following operation the patient was restless; complains of pain in right side of head and neck. Some twitching of extremities. Profuse sweating and chilly sensations at intervals. On July 27th, temperature maximum 103.2, pulse 128, respirations 26; on July 28th, temperature 104.4 maximum; continued daily high temperature, septic type. Continually complains of right neck and right head. Spinal fluid negative. On July 30th, blood culture was positive; "many strep. found." Developed pain in left shoulder and chest. General examination negative. Eye grounds negative. Nervous system negative.

Diagnosis: Right lateral sinus phlebitis. General septicemia.

Treatment: Because of financial difficulties, patient's parents

insisted on removing her to charity hospital. Report from surgeon in charge as follows:

"At the time of her admission she had a temperature of 102.8, W. B. C. 8,500, spinal cell count 7, and the spinal fluid was not under pressure. The Queckenstedt was negative, which we would expect when there is not complete thrombosis. At the time of the operation the lateral sinus wall was found to be markedly thickened and covered with dirty granulation tissue. The lateral sinus was blocked and the internal jugular ligated. She was seen by the Department of Medicine and a diagnosis of terminal septicemia was made. The neurologic findings were negative. The throat culture and blood Wassermann were both negative, and the blood culture was returned as streptococcus hemolyticus. The clinical course of her temperature was septic in type, going as high as 106, but never below 102. On the day of her death the W. B. C. was 21,500."

CONCLUSION.

Tenderness along the course of the jugular in the neck, with accompanying tenderness over the course of the vein in the skull, with headache on the same side, is a very valuable sign. All other causes of cervical and cephalic tenderness are first to be ruled out.

Rarely we have an epidemic of otitis in which the incidence of sinus complication is very high.

Each time transfusion of whole blood was done it seemed to either temporarily or permanently improve the patient's condition.

Abstracts of Current Articles.

Aspiration in the Treatment of Acute Suppurative Otitis Media. (*L'aspiration dans le Traitement de l'otite moyenne aiguë suppurée.*)

G. A. Weill, M. D. (Paris).

The author uses three sizes of short glass tubes, with a short piece of rubber tubing to fit into the external meatus, and an ordinary rubber medicine-dropper bulb to supply the suction. After preliminary cleansing of the canal, the device is fitted into the ear with the bulb compressed, and left in place one-half to one hour. This treatment is repeated once to three times a day. Irrigation of the ear is rarely needed. F.

The Sympathetic System as Related to the Caloric Test. (Mécanisme de production de l'épreuve Calorique de Bárány—Rôle du Système Végétatif.)

G. Buzoiany, M. D. (Bucharest), Arch. Int. Lar., 36:680, June, 1930.

By experimental suppression of the parasympathetic through intravenous injection of atropin, and through study of an individual whose cervical sympathetic ganglia were surgically removed (for epilepsy), the author concludes that sympathicotonus—probably through vasomotor stimulation—augments caloric irritability. This is notable in hyperthyroidism. Vasotonic persons present diminished caloric reactions. Herein lies explanation of many irregularities in the reactions of so-called neurotic or of tired, overworked people; the tonus of the labyrinthine circulation may be at fault. F.

Cancer of the Larynx. (El Cancer de la Laringe.)

Prof. Dr. A. G. Tapia (Madrid), Rev. Esp. y Am. de L., O. and R., 21: 289, July, 1930.

In Tapia's clinics, the incidence of laryngeal cancer reaches 3 per cent; and the percentage he now sees annually is double that seen in the early part of the twenty-eight year period surveyed. There is but one case in woman to 165 in man, much lower proportionally than in England, Poland and Germany. He considers tobacco the most important etiologic factor. Four of his six

female cases smoked to excess, as did fourteen other women cited by Barajas and Segni.

Tapia is opposed to biopsy unless the patient is willing to accept radical operation as a result of the findings. He mentions two cases in which tuberculosis and cancer were found together through biopsy. Pathologic findings must be confirmed by clinical observation before operation is decreed.

Operations performed, depending on extent and location of growth, include:

Laryngofissure—Twenty-two, no mortality, 14 cured, 7 requiring further operation; 1 not heard from:

Subhyoid pharyngotomy (when epiglottis involved)—Five, all recurred.

Lateral hemilaryngectomy—Sixteen, 1 death, 7 cured, 7 recurrences. (He considers this the most difficult excision operation.)

Anterior hemilaryngectomy—Four (since 1924), all cured.

This procedure in two cases included resection of the anterior third of the cricoid. The first skin incision is vertical, with a horizontal cut above the larynx and below the cricoid. This I-shaped incision in the skin is used to turn in over the stumps of the thyroid cartilage, leaving a large oblong opening which is packed. In two or three weeks this fistula is closed by sliding skin-flaps.

Total laryngectomy is indicated when the tumor occupies half the larynx and crosses the median line either in front, or especially behind; or

If limited to one side, when found in persons under 35 years, or when a basal-celled growth is present;

If it be adenocarcinoma;

If the epiglottis be extensively involved, whether the cords are or not;

If the lower half of the epiglottis is involved with extension to the cords or vice versa;

When it begins to extend outside the larynx, even though tongue and pharynx are not involved.

Persons older than 65, or suffering from heart or kidney disorders, tuberculosis, etc., he does not attempt. Existence of glandular metastasis, if not very large, he disregards, with occasional success in removal of such masses. The technic of Gluck is advised.

Of 190 cases, he had 12 operative deaths and recurrence in 35 per cent.

He advises radiotherapy in all cases where operation is refused or is inadvisable and as an adjunct to surgical removal. F.

Auricular Protheses. (La Prothèse Auriculaire.)

H. Chenet (Paris), Ann. d'Oto-Lar., 1:1, Jan., 1931.

Discarding celluloid and rubber devices held on by a spring across the vertex, Chenet recommends a master-cast modeled to imitate the opposite auricle, from which mold the patient can cast his own protheses from the author's improved gelatin mixture, every four to five days as needed. The present formula for this paste, which is colored with carmine, ocher and white to the proper flesh tint, and which no longer shrinks and checks during wear, is as follows:

Gelatin	100	in addition to that needed for soaking up the gelatin
Glycerin	150	
Gum arabic	75	in just enough water to dissolve
Alcohol	50	

This paste is melted in a water bath and poured into the mold, where it cools in a few minutes, and may be immediately worn, day and night. A little glycerin and alcohol may be added when the paste gets too stiff to melt readily. F.

Indications for Treatment in Otogenic Infectious Labyrinthitis.

(Indications thérapeutiques dans les labyrinthites infectieuses otogènes.)

J. Ramadier (Paris), Ann. d'Oto-Lar., 1:42, Jan., 1931.

This important study classifies the various labyrinthine symptoms arising from purulent otitis with much care. The author feels that it is rarely proper to open the labyrinth to prevent brain and meningeal complications. Only complete, delayed infectious labyrinthitis after acute otitis or total acute labyrinthitis

complicating chronic otitis, or total chronic labyrinthitis with manifest symptoms, suggest this rare operative procedure.

He feels that early and extensive mastoidectomy will generally avoid stirring up such latent labyrinthine foci as might lead to the above serious complications.

Operation on the labyrinth in late acute cases or in severe chronic otitis bears a very dark prognosis.

On the contrary, destruction of the labyrinth for the relief of severe vertigo is a harmless and very satisfactory operation, in cases where very little hearing remains in the ear to be operated upon. F.

Peritonsillar Abscesses. An Experimental Study. (Les abcès péri-amygdaliens.)

Prof. G. Canuyt (Strasbourg), Ann. d'Oto-Lar., 1:61, Jan., 1931.

Using fresh cadavers, and reporting one postmortem, Canuyt found that injected materials as well as the abscess in his fatal case were found in the extracapsular separable peritonsillar space, especially in the upper third of the fossa. On the living, peritonsillar abscesses were aspirated and the cavity refilled by five to ten cc. of lipiodol; front, lateral and basal radiographs were made. With variations depending on previous adhesions, they all resembled a hood or dome above and lateral to the tonsillar fossa, occasionally extending toward the pharynx and posterior pillar; never into the palatine recess or outward into the deeper planes of the neck.

Removal of the tonsil during acute attacks disclosed an intact pharyngeal wall, even when heavy masses of adhesions and scars were present; and he sees no disadvantage in such operations.

Canuyt recommends local anesthesia of the anterior pillar and makes a long incision, paralleling the outer surface of the tonsil, toward the last molar tooth. He finds the gaping wound made by the electric knife advantageous. The knife point and subsequent dull dissection must be directed inward along the tonsil instead of outward toward the muscles and great vessels. F.

The two leading French journals of otolaryngology, *Archives Internationales de L. O. R.*, and *Annales des Maladies de l'O. L. N.*, have been merged with the January issue into *Les Annales d'Oto-Laryngologie*, with the combined editorship of Lemaitre and Hautant. The publisher is Masson & Cie., and the new journal's format is very attractive.

The date of the next International Congress of Oto-Rhino-Laryngology has been set for September 27th to 30th, 1932, under the presidency of Prof. A. G. Tapia, in Madrid.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL
SOCIETY.

Meeting of Monday, December 1, 1930.

THE PRESIDENT, DR. GEORGE J. DENNIS, IN THE CHAIR.

SYMPOSIUM: SPHENOIDAL SINUSITIS AS A CAUSE OF
OPTIC NEURITIS.

I. Anatomy: The Relation of the Orbit and Its Contents to the Paranasal Sinuses (Illustrated with Lantern Slides).

B. J. ANSON, M. D.,

Associate Professor of Anatomy, Northwestern University
Medical School.

(ABSTRACT.)

The cancellous diploic tissue within four of the seven bones of the orbital cavity is typically replaced by epithelium-lined pneumatic spaces which retain, through minute apertures, their continuity with the nasal mucous membrane. Any or all these accessory nasal chambers may extend beyond the customary limits, to virtually surround, as a group, the entire orbit. In such instances the capacious maxillary sinus may extend beyond its typical position beneath the orbital floor and encroach upon the medial and lateral walls; the frontal sinus may invade the roof to an unusual degree, and matched in its invasion by the ethmoid cells, may extend backward to the very apex of the orbit; here an enlargement of the sphenoid sinus may increase the latter's relation to the cranial and canalicular portions of the optic nerve.

The integrity of the orbit and its contents is constantly menaced by the proximity of these spaces, which, as they lie adjacent to the orbit, may behave as the intermediaries by which a nasal infection becomes orbital.

Such an infection, upon successfully eroding the thin retaining wall of bone, next encounters the periosteal sheath which is but loosely attached to the walls of the orbit. Within this funnel-shaped sheath, and embedded in fatty areolar tissue, are such

important accessory organs of the eye as the ocular muscles, their nerves, the ophthalmic vessels and the optic nerve. The latter is additionally protected by coverings of meningeal origin, which, as they extend forward, become continuous with the protective coats of the eyeball.

II. Neurology: The Relation Between the Involvement of Sphenoidal Sinuses and Optic Neuritis.

GEORGE W. HALL, M. D.

(ABSTRACT.)

For the past several years I have been concerned about the relation between multiple sclerosis and retrobulbar neuritis. I will confine my remarks chiefly to that form of neuritis in discussing this topic. It is my opinion that the early cases of retrobulbar neuritis are seen by the ophthalmologist rather than the rhinologist or neurologist. The ophthalmologist finds on examination that the patients have a central scotoma with an enlarged blind spot, etc., and in many instances they do not give sufficient consideration to the relation between those symptoms and multiple sclerosis. In the majority of those cases our observations during the past few years have taught us to diagnose multiple sclerosis when central scotomata, the loss of abdominal reflexes, and a possible Babinski on one side or the other are present. Unfortunately, the neurologist does not come in contact with the majority of such cases during the early period. Most textbooks describe the symptoms of a classical picture of multiple sclerosis, such as bitemporal atrophy, nystagmus, scanning speech, intention tremor, bladder disturbance, etc., whereas we well know at the present time that such symptoms when present represent a rather advanced case of multiple sclerosis. I have no doubt that multiple sclerosis is of bacterial origin, the infection, no doubt, coming through the blood stream. It may originate from foci in different parts of the body. In determining the causal relation of the infection of the sphenoid sinuses to retrobulbar neuritis one must take into consideration the experience of men who have seen a large amount of clinical material in order to compute the relative relation between the source of infection and the disease itself. I know of no one better than Von Hippel, who has for

many years protested against the assumption that retrobulbar neuritis is frequently caused by sinus disease. He reports seventy cases seen during the past fourteen years with forty-eight cures and only one nasal operation. In nineteen of these cases the etiology was multiple sclerosis; in twenty-seven cases the etiology was unexplained. He comments upon these figures by stating that if operation had been performed in the early stages such statistics would have been regarded as highly favorable to operative measures. He further states that forty-one of the cases in which the etiology was never clear were under observation for several years, and many of them corresponded in their course to that of multiple sclerosis. Wilfred Harris¹ states that retrobulbar neuritis is frequently caused by a patch of disseminated sclerosis invading the nerve trunk, producing more or less sudden blindness, which may be mistaken for hysterical amblyopia. He further states that retrobulbar neuritis may also be due to a variety of toxic causes, of which tobacco amblyopia is well known. Central scotoma has also been described, according to this author, as following the optic atrophy of *tabes dorsalis*, which sometimes leads on to blindness, and which occurs rather seldom in cases of disseminated sclerosis. In *tabes dorsalis* the lesion is a parenchymatous degeneration of the nerve fibers, while in disseminated sclerosis the lesion is primarily inflammatory in character.

Friedenwald² states that the optic nerve and its path through the optic foramen is brought close to the sphenoid sinus. It has been shown that considerable variation exists in the various structures of this region. It has also been claimed that certain cases of retrobulbar neuritis are greatly helped by the treatment of concomitant nasal sinus disease, usually involving the sphenoid or ethmoid sinuses. He is of the opinion, however, that a causal relationship between these two diseases has never been proven. On page 64 of the same report he further states that a very large proportion of the retrobulbar neuritis is due to multiple sclerosis, the typical lesion of which has been demonstrated in the optic nerve. He also mentions the fact that such involvement may be of luetic origin. In the discussion of a paper by Robert J. Hunter,³ Vail states that he has never seen a case of retrobulbar neuritis of nasal origin in a child. George E. Brown,

discussing the same paper, in reporting sixty cases, stated that the primary focus occurred in the tonsils in 17 per cent, in the teeth in 14 per cent, in the antrum in 2 per cent and in but six cases was the ethmoid involved.

Leon White⁴ stated that the absence of visible pathology in the sinuses in cases of retrobulbar neuritis caused him to look for other foci of infection. He was of the opinion that invasion is through the blood stream and that the accessory sinuses play only an insignificant part as the primary focus of infection. He further stated that, in his opinion, most of the foci are found in the teeth and tonsils, and among the sinuses the antrum is the most frequent seat of infection. In 80 per cent of his series the patients had infected teeth or tonsils, whereas sinusitis was present in only a very small percentage of the cases. On the other hand, I am frank to say one can find many authorities who are just as firmly convinced that sinus infection is, in the majority of cases, the cause of retrobulbar neuritis. For instance, Glas-scheib⁵ states that there is a definite connection between sinus disease and retrobulbar neuritis, and he is of the opinion that operators very frequently overlook the presence of sinus disease at the time of operation. He goes on to say that a transudate of the ethmoid and sphenoid cavity with formation of edema may be present. He quotes Weill as being opposed to such a theory. The latter authority concludes that rhinologists do not find retrobulbar neuritis in association with postsinusitis, and ophthalmologists do not find sinus disease in acute retrobulbar neuritis.

Langenbeck⁶ reported 176 cases of retrobulbar neuritis in which he found only 3.5 per cent were of nasal origin; 77 of the 176 cases had multiple sclerosis. Consequently, one cannot minimize the value of reports such as those I have mentioned, and considering their statistics at their face value, it appears to me that we must arrive at the conclusion that sinus disease is, relatively speaking, a rather infrequent etiologic factor in the production of retrobulbar neuritis; that retrobulbar neuritis most probably is secondary to foci of infection located in almost any part of the body leading up to rather diffuse changes in the central nervous system which actually correspond to those of multiple sclerosis, and that, after all, the relation between multiple sclerosis and

retrobulbar neuritis is probably of as frequent occurrence as all other causes combined.

I desire to make it plain, however, that I have no quarrel with either the rhinologists or the ophthalmologists, that I can only hope that the three specialties may in the future become more interested in this subject, and that we may consult one another more often than we have in the past, in order that much greater service can be rendered the patient. I believe that if we can diagnose the early diffuse changes in the central nervous system at the time or shortly after the period when the ophthalmologist first sees these cases of central scotoma we may be able to institute treatment whereby the progress of the disease can be checked and the patients may not become completely invalidated.

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III. Eye Symptoms.

GEORGE F. SUKER, M. D.

(ABSTRACT.)

There are two clinical types of optic nerve involvement: optic neuritis and retrobulbar optic neuritis. In the former, the papillo-macular fibers are seldom, if ever, implicated but in the latter the involvement of this bundle is the characteristic feature. Either one may be due to toxins or constitutional lesions. In optic neuritis the objective clinical findings are usually limited to the optic disc at the outset, and subsequently descend into the nerve. While in retrobulbar optic neuritis very few, in fact, only two, are first seen (in a triangular temporal area of the disc), subsequently the whole disc may become involved.

The prognosis, with few exceptions, is decidedly more favorable in retrobulbar neuritis than in optic neuritis.

Aside from tumors, congenital anomalies of the sphenoid, associated with disease and injuries of the sphenoid sinus causing optic neuritis, choked disc and field changes, sphenoid sinusitis very seldom causes a retrobulbar optic neuritis.

No doubt retrobulbar neuritis can be caused by affections of the sphenoid, but it is so rare that conclusive evidence is not found, either clinically or pathologically, so that one must be extremely careful in dogmatically stating that a large percentage of retrobulbar neuritis is of sphenoid origin. No doubt, particularly if so-called pathologic anatomic variations of the sphenoid exist which will permit of the implication of the retrobulbar nerve and a septic condition exists in the sphenoid, it is possible for a retrobulbar neuritis to develop.

By far the largest percentage of retrobulbar neuritis is self-limited and the nerve recovers without any sphenoid sinus surgery. Most often when no direct toxicity, such as alcohol and other poisons, injuries and heredity, including syphilis, can be elicited, the retrobulbar neuritis is a precursor of multiple sclerosis.

Very frequently no active pathology can be found in the sphenoid with an added retrobulbar optic neuritis, and the removal of redundant sphenoid tissue followed by a disappearance of the neuritis does not constitute a causal relationship.

An enlarged blind spot of itself is not a retrobulbar optic neuritis, even in the presence of a sphenoid complication. An enlarged blind spot is not necessarily associated with a central scotoma, neuritis or visual reduction. Any pressure upon the retrobulbar nerve from any cause can produce an enlarged blind spot, but if an enlarged blind spot is associated with a central scotoma, visual reduction and field interference, and a temporal pallor of the optic disc, then there is a true retrobulbar optic neuritis. These findings one seldom obtains when suspicion is directed to the sphenoid sinus. Before it can be definitely stated that the sphenoiditis is the etiologic factor of a retrobulbar optic neuritis, it is obligatory to eliminate the following more potent and usual causes:

1. Acute myelitis
2. Multiple sclerosis
3. Leber's hereditary optic atrophy
4. Tobacco-alcohol amblyopia
5. Diabetic amblyopia
6. Neuritis from a gravid uterus

7. Lactation
8. Sinus infections.
9. Infections and intoxications
10. Syphilis (cerebrospinal)
11. Tuberculosis
12. Typhus
13. Erysipelas
14. Beri-beri
15. Influenza
16. Mumps
17. Pneumonia
18. Angina
19. Meningitis
20. Encephalitis
21. Smallpox
22. Measles
23. Pertussis
24. Vaccination
25. Bright's disease
26. Severe burns (hands and face)
27. Carcinomatosis
28. Frontal lobe tumors
29. Chiasmal lesion—posterior angle
30. Methyl alcohol
31. Quinin, etc.
32. Tabes
33. Lesions in the vicinity of the posterior angle
of the chiasm
34. Third ventricle lesions
35. Brain abscess
36. Hyperthyroidism

By no means do these complete the category.

So frequently do remissions occur in retrobulbar optic neuritis, due to the above conditions, that should a sphenoid be faulty in addition, and an apparent improvement follow sphenoid intervention, the latter cannot really be called the true etiologic factor. The retrobulbar optic neuritis is quite often the forerunner by months and years of some of these systemic dyscrasias.

The papillomacular fibers of the optic nerve are very susceptible to toxins, especially the retrobulbar section of this bundle, because they are more exposed in the nerve near the chiasm than anywhere else in the nerve or tract. It is not uncommon for basal lesions or toxins to produce enlarged blind spots with central scotomata and consequent field and visual disturbances, with and without evident temporal pallor of the disc, thus giving rise to a picture of retrobulbar optic neuritis.

True it is that given a sphenoid with dehiscences and active infection, a retrobulbar neuritis can ensue. But this is an anatomic abnormality, allowing the passage of the toxins into the vicinity of the retrobulbar nerve. These dehiscences are not as frequent as the supposed retrobulbar optic neuritis of sphenoid origin.

Though the anatomic relation between the sphenoid wall and the retrobulbar nerve may be very intimate, the dense periosteum lining the sphenoid and the sheath of the nerve will scarcely permit of easy or ready access to the papillomacular fibers of any sphenoid infection. Investigators in this field have rarely reported retrobulbar optic neuritis when a portion of the retrobulbar nerve was completely enveloped within the sinus and there existed a so-called sinusitis.

An optic neuritis can follow a sinus infection. In such instances there are inflammatory manifestations on and in the disc unassociated with a central color scotoma and temporal pallor. But an optic neuritis and retrobulbar optic neuritis are not one and the same lesion. The latter seldom gives any visible disc changes other than a temporal pallor, excepting in the latter stages when a neuritis may be engrafted, too late for any sphenoid interference to be of any benefit should suspicion be directed against it. There are so many other potent underlying lesions than sphenoid disease causing retrobulbar neuritis that the latter can only be exceptional and incidental.

The classic triangular temporal pallor of the disc in retrobulbar optic neuritis I have not seen in any case supposed to have been due to so-called sphenoiditis.

The diagnostic clinical features of retrobulbar optic neuritis are:

1. Triangular pallor of the temporal quadrant of the disc, in which area the capillaries are practically invisible or markedly attenuated.

2. A central scotoma for red and green; at times for other colors also.

3. Pain upon pressing the globe into the orbit.

4. More or less pain in ocular movements.

5. An enlarged blind spot, not common.

6. A possible visual field infringement.

7. Early reduction in visual acuity.

8. The disc contour is not disturbed, excepting when a disc neuritis supervenes.

9. No vessel changes in disc except in temporal pallor area, unless there is an engrafted disc neuritis.

10. The physiologic cup of the disc is scarcely ever obliterated.

11. No adjacent minute hemorrhages or exudates.

12. Choked disc appearance usually absent.

In an optic neuritis the diagnostic points are:

1. The disc contour is apt to merge with the adjacent retina.

2. The physiologic cup is often obliterated.

3. The vessel contour is disturbed.

4. No temporal pallor (triangular).

5. The whole disc is distinctly inflamed.

6. Minute hemorrhages on disc or adjacent to same may occur.

7. Not unusual to have some exudate on disc or in immediate vicinity.

8. Visual acuity frequently is reduced.

9. Field infringement is rather common.

10. Seldom if ever pain of any kind, either upon rotation or of pressure on globe.

11. Disc not uncommonly so swollen as to be mistaken for a choked disc.

12. Blind spot usually enlarged.

Judging from my experience, retrobulbar optic neuritis from a sphenoid lesion is an extreme rarity. However, so-called optic neuritis, with the characteristic enlargement of the blind spot, is not very uncommon. In all sinus diseases we should carefully

examine the blind spot, as it often is an early manifestation of an impending toxic optic neuritis.

A New Method of Demonstrating the Relation of Sphenoidal Sinus and the Optic Nerve.

HARRIS H. VAIL, M. D.,

CINCINNATI.

(ABSTRACT.)

A brief résumé of the literature quoted by Canuyt, Ramadeier and Velter would seem to show that for several centuries retrobulbar optic neuritis of nasal sinus origin has been recognized.

The pathologic studies of Prof. Herzog of Innsbruck are quoted, and his belief emphasized, that they will explain in every way the mechanism of the production of retrobulbar optic neuritis from posterior nasal sinusitis.

Statistics of various authors are quoted to show that the incidence of retrobulbar optic neuritis of nasal sinus origin varies according to the particular author's acceptance or denial of a nasal sinus etiology of retrobulbar optic neuritis.

A series of fifteen cases of retrobulbar optic neuritis of nasal origin seen by the author is tabulated.

An important observation is made, that where the onset of blindness was acute and could be definitely dated, it always was noticed after awakening from sleep. The position of the head in sleeping would naturally favor the gravitation of infection to the upper outer angle of the sphenoid sinus where the optic nerve is in closest relation.

Statistics are quoted to show that the eye symptoms constitute an early symptom of multiple sclerosis in only 14 per cent of the cases, and the opinion is stressed that the virus of multiple sclerosis may gain entrance into the central nervous system from the sphenoid sinus in the same way that organisms have been shown to do. If this is a fact, it immediately would indicate that early surgery to the posterior sinuses should be done upon cases of multiple sclerosis.

A new method is described of roentgenographic visualization of the relations between the optic canal and the sphenoid sinus by means of filling the sinus with radiopaque oil and so placing

the patient's head that the upper angle of the sinus is dependent. Thus, the radiopaque oil comes into contact with the boundary of the sphenoid sinus where it is most closely related to the optic nerve. In no other position can this be shown.

DISCUSSION.

DR. HARRY GRADLE: I have a few very tender corns in this respect that have been trodden upon tonight. I feel that there are other phases to the subject than a mere blanket denial that such a thing as rhinogenic retrobulbar neuritis does not exist. I am certain that it does. It is perhaps not as frequent a cause of retrobulbar neuritis as some believe, nor is it as infrequent as others believe.

Optic neuritis *per se* is not an evidence of sinus disease. A toxemia from sinus disease will seldom produce an optic nerve involvement. The involvement is that of the retrobulbar type. (Illustrating on blackboard.) If we consider the orbit and the canal through which the optic nerve passes, we will find a few important structures for consideration. As the posterior mesial wall of the orbit is formed in part by the lateral wall of the sphenoid or ethmoid, the separation between these cavities and the orbit is a very thin plate of bone. Lining the wall of the orbit is the periosteum, which at the optic canal fuses with the dura covering the optic nerve, so that any toxin passing through the plate of the orbit and involving the periosteum will involve the dura in this rigid canal. When we consider the structures that lie from here to the orbit we must consider first the vascular portion of the optic nerve, then the avascular portion, and then the intracanalicular portion. If the offending agent involves any portion of the nerve posterior to the entrance of the central artery of the retina, that involvement is manifested only by a study of the visual fields of that patient primarily. The avascular portion between the entrance of the central artery and the intracanalicular portion is not likely to be involved.

In cross section of the nerve in the intracanalicular part, the fibers that come from the retina immediately around the optic nerve in the eye are found in the periphery. Lying in the central portion we will find a horseshoe-shaped bundle containing those

fibers relating to the central vision. The remainder of this portion is filled with nerves coming from the periphery of the retina. The first portion to suffer are the fibers that come from the blind spot, and the first indication of a retrobulbar neuritis is not central scotoma or pallor of the disc, but enlargement of the blind spot which can be measured accurately. When the process has advanced so far as to produce a central scotoma it has then involved the nerve in the central portion and we have a much more serious condition. There are undoubtedly many, many cases of retrobulbar neuritis of rhinologic origin that are due to a blockage of the aperture with reversal of the drainage. Many of those cases are overlooked entirely, without doubt. Many of the cases are overlooked until the process has progressed to the formation of the central scotomata, but by careful search of the blind spot you will be able to find many cases of retrobulbar neuritis presenting the symptoms Dr. Suker mentioned to a mild degree, presenting enlargement of the blind spot which will progress gradually to scotoma for colors and then a complete central scotoma.

It is equally true, as Dr. Hall said, that many of the cases due to multiple sclerosis will improve upon ventilation of the sinus. It is also true that many of the cases that in the beginning we were inclined to attribute to sinus origin will finally prove to be multiple sclerosis cases. I have been looking for those cases since 1914 and have a fairly good record of such cases, and if those patients were to develop multiple sclerosis they would have done so before this time.

One other point, I believe, is that the retrobulbar neuritis that is manifested primarily by central scotoma is due to lesions lying in the central area of the optic nerve. Those lesions lying in this central area may well produce as a primary lesion a central scotoma and temporal pallor of the disc.

I wish to go on record as believing that there is a definite retrobulbar neuritis due to rhinogenic origin, which can be relieved if properly treated.

DR. EDWIN MCGINNIS: Of the lessons that can be learned from this meeting the lesson of cooperation is paramount. This subject is of great interest, and I think there is something to the

infection in the nasal sinuses. Dr. Hall emphasized the nervous side of the question, and I think if we go into it thoroughly, with careful check-ups, we will find that many patients have nervous upsets and many have sinus infection as well. It is hard to prove experimentally that one can take some bugs out of the nose and produce optic neuritis in an animal, but if we could do that it would help in our determining what the etiology is. The best thing we can do is to make a diagnosis, get the neurologic findings, the ophthalmologic findings and all work together. It seems to me that if we have a patient who has gone blind, who has been examined by a competent ophthalmologist, who has been gone over by a neurologist and the neurologic side investigated, if you operate the nasal cells and in twenty-four hours vision improves and tension improves you can see that probably the infected cell had something to do with it. If you also see the patient return in a few months who has had a cold and vision is dim, and you clean up the nose and vision improves, you will say that probably the infected cell had something to do with the condition.

I had the pleasure of going down to see White and followed his three stages of focal infection. His first stage was ethmoiditis and sphenoiditis, and I saw several of his operations and followed them. In the second stage he was investigating the size of the optic foramen and was much sold on the idea that if an individual had a small optic foramen, if they developed infection of the sphenoid they were likely to get optic neuritis. I heard him read his paper and listened to the discussion. It was brought out that if an individual has a small foramen he has a small optic nerve. When I saw him again in his third stage, he was then all off the size of the optic foramen and all for infection of the posterior ethmoid cells and of the sphenoid. He was all for removing the tonsils and teeth and was sure that the trouble was due to infected teeth or tonsils. Unfortunately, he died. I think he would have enjoyed listening tonight to Dr. Vail's paper, for Dr. Vail has helped out in the relationship of the sphenoid cell to the sinus.

I do not wish to go away with the idea that if we see a patient with a retrobulbar neuritis that as soon as you look at the sphenoid

noid cell you have to find an infection in there. I think the best thing to do is to examine the nose and see if there is an infection. Very often we will find an infection in the anterior cell group. I have one patient who has an infected antrum. I got so enthusiastic about it that last spring I wrote to the chairman of the Section on Otolaryngology saying that I would prepare a paper on the intranasal findings in certain eye lesions, and I was going to bring out the findings in the cases I have seen at the hospital. I am sorry to say that in only one of the cases did we have a sphenoid infection. All the cases I have seen of eye involvement have been referred to me by the neurologic surgeons or the ophthalmologic surgeons, and in many cases they have been of great assistance in helping out in the care of the patient. It has been surprising to have an ophthalmologist call up and ask what the findings were, was the sinus full of pus, or what was the condition. I remember one case that was diagnosed as retrobulbar neuritis on Dr. Bassoe's service. Dr. Bassoe made the notation on the chart that this man, he felt sure, had an ethmoid infection which had gone up along the sinus and involved the nerves. He wanted me to open up the ethmoid cells and see what we could get out. I did this and found only an inflammation on the right. The patient was soon able to see the chandelier in the center of the room and in three weeks could see perfectly. That was worth while, for he was a country doctor and needed his eyes to make a living. A few months later he returned after he had had a cold and the vision again was involved. I cocaineized the cells and he was soon able to return to his work. I feel that we cannot take any stock in the various observations the rhinologists have where they are of various different stages. The older men do not believe in operations on the nasal cells and the young are perhaps too enthusiastic. I remember one case in a prominent man who had a bilateral optic opacity. He went through the Northwestern group, where someone examined his nose and throat and reported the nasal findings as negative. He had gone through the Rush medical group, and a nose and throat man had examined him and had given a negative report as to infection of the nasal cells. When I saw him both sides of the nose were filled with pus and he had a bilateral nasal dis-

charge. I asked him if he had this condition all the time and he said "Yes." We took some pictures and found an edematous nasal membrane but were unable to persuade him to let us do anything to his nose, because the two medical groups had given negative reports. I think if someone could have opened up his nasal cells he would not have lost his vision.

Let us pass to multiple sclerosis. A patient had a nasal discharge which was more evident on the left side. She was referred by Dr. Wilder with a diagnosis of retrobulbar neuritis. My diagnosis was ethmoiditis and I opened both ethmoid cells back to the sphenoid. The patient went on for six to eight weeks without improvement of the eye condition. After she returned home her vision came back but she developed multiple sclerosis. She was in bed for some time but the multiple sclerosis cleared up. She never saw me during her stay in the hospital and she returned to Chicago later to see what I looked like. She then had a vision of 20/20 and was very grateful for what I had done.

I was very glad to see Dr. Gradle's drawings and hear his remarks on the possibility of having infection in the optic nerve just back of the eyeball, for I have seen patients with just an optic neuritis and not a posterior cell invasion. I have operated upon about 1,200 ethmoid cases in the last sixteen or eighteen years, and I do it by a procedure by which I can watch each cell as I open it. It is surprising how few of these cells look as though they had bacteria in the membrane.

I think we have had a great lesson in this meeting, in that we must use teamwork, and if we can work together we certainly will get somewhere.

DR. ROBERT BLUE: A week ago today I was consulted by a young lady who gave the following interesting history. Two weeks before I saw her she underwent a laparotomy. While she was in the hospital she developed a disturbance of vision which did not attract her attention, but upon her return home the visual defect attracted her attention in the following manner: She had a lamp with a red globe and when she closed her right eye she could not see the red globe when looking directly at it, but upon looking either above or below it she could see it. She stated that she had a similar attack in the other eye about nine years before.

At that time she had been in Cincinnati and had consulted Dr. Derrick Vail, who made a diagnosis of retrobulbar neuritis and referred her to Dr. Harris H. Vail. Dr. Vail opened her sphenoid and posterior ethmoid cells, and a week later her vision had returned to normal. That attack was in the right eye, and this time the left eye was affected. Her vision consisted of ability to count fingers at two and a half feet; there was definite central scotoma, relative. The only other symptom was that on rotating the eyes there was a sensation of pressure. The left eye was a trifle more sensitive than the right, but there was no actual pain, just a slight discomfort. I asked her if she could see well with that eye before, and she told me it had always been a poor eye and had been crossed when she was a child. I made a diagnosis of probable retrobulbar neuritis in an amblyopic eye. At the request of her surgeon, I referred her to Dr. Bookwalter, who made a roentgenogram and found the sphenoid and ethmoids clear. I saw her again last Saturday, and the vision had improved in that eye to the ability to count fingers at about twenty feet. The same day I received from Dr. Vail a letter in which he gave the findings of nine years before. The vision in the right eye at that time was an ability to count fingers at a few feet. About twelve days later the vision was 20/30. In the left eye the vision on Saturday was ability to count fingers at about twenty feet, which approaches the normal vision of 20/200 which she had nine years before.

This patient gave one rather interesting point in the history, to the effect that some years before she had taken a general ether anesthetic and at that time noticed some visual disturbances. They did not, however, attract her attention sufficiently to call the attention of her physician to the disturbance, but she thought the attack was similar to the present one. I am inclined to give a little more than average weight to her history because it was all borne out most accurately by the report I received from Dr. Vail. Her observations have been accurate all the way through. We have not made a diagnosis as to the etiology in this present attack, but we have to consider sinus infection, which we apparently have ruled out. We must consider also the ether as being a possible cause, although I have never had a case of retrobulbar

neuritis due to ether under my observation. Also, I presume we shall have to refer the patient to Dr. Hall to discover whether or not the real underlying cause is a multiple sclerosis.

DR. JOHN A. CAVANAUGH: The sphenoid question has been of great interest to me, as I have been studying its various phases for a long while. About eight years ago I conferred with Dr. Hubeny on the possibility of detecting the relation of the optic nerve to the sphenoid sinus, according to the principle of locating a foreign body in the eye.

Dr. Hubeny did not think this could be done, since we have no fixed anatomic relations such as are used for detecting foreign bodies in the eye. However, we made a number of roentgenograms, which showed the sphenoid sinus overlapping the optic foramen. The relation varied in the different subjects, which we attributed to the development of the sinus.

The fluids used have been varied, bismuth in buttermilk and in malted milk, lipiodol and brominol. I do not agree with Dr. Vail's remarks on brominol. There are two types of the drug, light and heavy, and the type is selected according to the picture desired.

We have much to learn regarding the sphenoid sinus. Certain cases have localized areas of involvement that are hard to diagnose and it is difficult to get postmortem material.

DR. WALTER H. THEOBALD: After hearing the first part of this symposium I thought retrobulbar neuritis of accessory sinus origin had taken a tailspin, but the work which Dr. Vail has presented is well worth while. He has given us something more definite upon which to base judgment for diagnosis. He states that the sinuses are injected and if the opening is not exposed and accessible he makes an opening into the sphenoid. By doing that he does not tell us whether or not a sinus has normal drainage. Dr. Gradle pointed out that a blocked sinus would cause pressure or retention, and therefore Dr. Vail is basing his information upon thickened membranes only and not upon retention of secretions or their delayed drainage.

Illustrating this point, I will briefly review a case I saw with Dr. Gradle in which we used the method so well brought out by Dr. Proetz, namely, the introduction of radiopaques into the posterior sinuses by negative pressure. In this case the patient, a

woman, aged 30, was rapidly going blind in one eye. She had retrobulbar neuritis. Roentgenograms were made by injecting lipiodol with negative pressure. On the affected side the sphenoid did not take up any of the oil and we therefore concluded that this sinus was blocked. Whether there was a thickened mucous membrane or not was not of importance; the sphenoid was completely blocked. She was operated upon and the result, as in many of the cases cited this evening, was that vision came back to counting fingers on the third day and to reading a newspaper inside of a week.

DR. HARRIS H. VAIL, Cincinnati (closing): I wish to thank the gentlemen for their kind discussion.

Replying to Dr. Cavanaugh's question, the brominol used in the one case was that which a salesman left in my office for trial and I do not know whether it was the light or the heavy preparation.

The point brought out by Dr. Theobald is a good one. Dr. Proetz's suction displacement method is an excellent means of filling the posterior sinuses, but if I remember correctly his interpretations are based upon the emptying time of the sphenoid sinus under study. In dealing with the study of the sphenoid sinus in a suspected case of retrobulbar optic neuritis we are usually dealing with an acute infection which has often subsided by the time we see the patient, so that I believe in these cases it is much more important to study the sphenoid sinus for filling defects rather than to study it from the standpoint of its emptying time.

Herzog, as I mentioned in the article, found cases of acute sinusitis where the mucous membrane upon microscopic examination showed no evidence of infection, yet there were definite signs of inflammation in the submucosa and marrow spaces, so that one would hardly expect to find very many cases of filling defects in the sphenoid sinus in cases of retrobulbar optic neuritis. However, one case of recurrent attacks of retrobulbar optic neuritis did show a slight thickening of the sphenoid sinus mucosa.

It is important to remember that the roentgenograms should be made with the patient's head in the vertical position, both for

anterior, posterior and lateral views, and since I have used the special angle to show the filling of the upper outer portion of the sphenoid I believe I can determine just how close the radiopaque substance comes to the optic canal.

When I am unable to get into the sphenoid sinus through the ostium I then puncture the front wall. It takes a good deal of time to carry out this method, as the case must be thoroughly cocaineized just as for an operation upon the sphenoid. There are some cases where on account of septal deviation or very large middle turbinates it is impossible to inject the sphenoid, but in every case in the series of suspected and true cases of retrobulbar optic neuritis which I have seen, it has been possible to inject the sphenoid either through the ostium or by a puncture of the anterior wall.

I enjoyed Dr. Hall's paper very much. The question of multiple sclerosis is, of course, extremely important and interesting, and one must bear in mind the fact that the cause of multiple sclerosis may get into the central nervous system by way of the posterior nasal sinuses. Whether any experimental work along these lines can be done with animals I do not know, but I think it would be rather difficult on account of the anatomic development of the sinuses in animals, particularly dogs.

In chronic nasal sinusitis there must be a barrier formed around the focus of infection in the sinus, but like barriers elsewhere in the body, there may be gaps in them. I believe more cases of retrobulbar optic neuritis are seen in acute sinus infections because, on account of the anatomic relationships, infection can involve the nerve before the patient has a chance to develop his defenses.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL
SOCIETY.

Regular Meeting, January 5, 1931.

THE PRESIDENT, DR. GEORGE DENNIS, IN THE CHAIR.

**Motion Picture Demonstration of Normal Interior Laryngeal Functions
Which Block the View of the Glottal Lips.**

G. OSCAR RUSSELL,

Director of Phonetics Laboratory, Ohio State University,
Columbus, Ohio.

(AUTHOR'S ABSTRACT.)

The pitch used by the subject during articulation radically affects the view of the vocal lips.

Very low pitches cause so-called "retracted epiglottis." The pulvinar is pulled towards the cartilages of Wrisberg, causing posterior anterior closure.

Excessively high pitched tones or "tight" or strident voices cause lateral interior larynx constriction, hiding all but the mere edge of vocal lips.

Some vowels expose a view of the full length vocal lips, others shut off the view progressively more and more.

The larynx interior closes (anterior posteriorly) more and more, thus impeding the view in a progressively increasing way as we pass through the vowel series:

i-peep; I-pip; e-pape; e-pep; ae-pap; ə-pup; a-pop; ɔ-paw; o-pope; u-poop.

The X-ray pictures published in my "Speech and Voice" show that the front cavity for "ee," (i) is hardly as big as one's finger, and it is therefore impossible to insert an ordinary laryngoscope through it for any kind of a view (as one can my "*nongag glottoskop*" or my "*fonofaryngoskop*") without enlarging that anterior buccal cavity more than the vowel will permit. If enlarged, the vowel changes to either:

First, usually the vowel "uh" (ə) in "pup." It is true part of the cords are then visible but they look dumpy. The anterior half remains hidden.

Second, or the vowel "ah" (a) in "pop" producing so-called "retracted epiglottis." No cords are visible.

Third, or at best something between (I) in "pip" and (e) in "pep," when more of the cords will be visible. In other words, roentgenograms of the vowel "ee" (i)—peep, show the largest pharynx and smallest mouth distension. Hence, if proper apparatus is available, that is the best vowel to use in order to avoid impediment of the vocal cord view.

A guttural voice closes the false cords and hides the vocal lips.

Gagging causes both posterior anterior and lateral closure of the larynx, completely shutting off view of the glottal lips; that is to say, a gagging or interrupted voicing closes the false cords above the vocal lips.

A cough closes the ventricular bands above the vocal lips, thus hiding them.

Clearing the throat closes the false cords as well as the vocal lips.

Hence, it is absolutely necessary that the subject articulate on his normal easy range speaking tones, with relaxed free quality, if an unimpeded view of the vocal cords throughout their whole length, and of the entire interior larynx is to be had.

DISCUSSION.

DR. LEO KALLEN: At the general sessions held by the American Society for the Study of Disorders of Speech last Wednesday afternoon, I had the pleasure of witnessing Dr. Russell's demonstration, which, I was given to understand, would be repeated at this meeting tonight. Having received no abstract of Dr. Russell's address, my remarks are in reference to the brief abstract published in the program of last Wednesday afternoon.

That intralaryngeal activity, *per se*, other factors supposedly being equal, exercises an influence on, or rather that it actually is one of the conditions on which quality and character of voice depends, is a fact that has been known for several decades, beginning with the classic attempt of Czermak, in 1860, to photograph laryngoscopic images, which were successful enough to depict the cords in phonation, the shape of the glottic chink, and the relation of the ventricular bands.

With the development of photographic technic, depiction of the interior of the larynx in its varying phases of function, made corresponding progress.

However, it is really only in the last two decades or less that technical skill has made it possible to depict the larynx with an ever decreasing quantum of imperfection.

We are indebted to Dr. Russell for a most beautiful demonstration of a moving picture of intralaryngeal activity synchronized with its vocal effect. This is an important achievement, not only because of a faithful portrayal of a process otherwise hidden, except to the eye of a phonendoscopic observer, but also because synchronization with sound offers a new method of study and instruction. Those of us who are concerned with the diagnosis and treatment of functional disturbances of voice are in need of more and more information concerning intralaryngeal processes in connection with vocalization. Dr. Russell's success in synchronizing these two in the form of a tonal motion picture is an important step in that direction.

DR. ELMER L. KENYON: Somewhat more than a century ago the present national movement for the education of the deaf in speech and lip reading was inaugurated. About twenty-five years ago a national movement for the educational treatment of disorders of speech in the hearing was likewise inaugurated. Last week the fourth national convention of the American Society for the Study of Disorders of Speech, an organization which is attempting to guide this newly developing movement, was held in Chicago. I speak of this, not because I am the president of that society, but because just as the deaf have had to be associated with the physician in order to do their best work, so this newer movement for the speech disorders of the hearing should have the sympathetic co-operation of the medical profession. I speak of this, also, to show why our guest speaker has been stimulated to carry on his remarkable investigations.

Before taking up the presentation of moving pictures of the larynx, I wish to call the attention of the society again to the electrically lighted laryngoscopes that have been developed by Professor Russell, and which were first demonstrated before this

society about a year ago. By its very nature the ordinary laryngoscope, excepting in certain patients, for the sounds "ah" (are) or "aw" (awed), cannot show the interlaryngeal movements without interference with free vocal cord action, and, besides, cannot show most of the sounds of the language at all. Killian was instrumental in bringing the larynx into direct view, but with practically complete suppression of movement. Dr. Russell, following with his mechanically acute mind the more basal work of his predecessors, has opened up for us in two ways an almost unhampered view of the interior of the larynx in action for any sound. These new electrically lighted laryngoscopes are of real value to the laryngologist, and will in time become a part of his armamentarium and of that of our medical schools. Also, these new instruments can be used equally well by nonlaryngologists who, for example, are interested in disorders of speech. I wish to add that Dr. Russell receives no personal profit from the sale of these instruments.

Now we are enabled to witness further fundamental progress in the disclosure of the interior of the larynx in action. These interior movements are disclosed in detail and with clearness as moving pictures. About twenty years ago, working from the standpoint of the extrinsic movements of the larynx, I was able to point out that each basal sound of the language must require detailed differences in the approximation of the vocal cords. It is now of great interest to me to have the accuracy of my observations proven by these moving pictures of the interior of the larynx in action.

The interpretation of the significance of the movements, as disclosed, will require much time and study. Exactly what movements serve to produce a certain type of sound, as to language significance, musical quality and pitch, must be gradually determined. This information will be a companion to the evidence of the sound itself, which we have always had.

But these facts, even together, cannot solve the problem of developing the artistic voice. That problem concerns the question of so controlling the musculature of speech production as to attain to any desired vocal result. Just how these pictures are to forward the solution of that problem is yet to be determined.

The complexity of the movements within the larynx, as just disclosed, is remarkable. How do we get them—how do we control the larynx? Did you ever think that the larynx gives the brain no knowledge of what is going on within itself? There is no way in which the brain can directly understand what is going on in the larynx except in the vocal result. In order to realize the complexity of the act of voice production, we must think of the movements of the extrinsic muscles of the larynx, which also are complicated, as well as the intrinsic muscles. These muscles, extrinsic and intrinsic, are controlled in action for voice production by an automatic cerebral mechanism, which acts indirectly in accordance with the conscious purpose of the individual to produce a definite sound. The problem of voice development consists in the determination of exactly how to gain control of these muscles in order to produce any particular vocal result at will. The moving pictures throw new light on how the larynx produces particular vocal sounds, but they do not solve the problem of how to control the vocal mechanism in order to attain to particular sound results. What they may ultimately contribute to this practical purpose is a matter for the future to decide.

As to the value of such pictures to the laryngologist in the future, it is now too early to prophesy. But that they may be developed into a diagnostic procedure of much importance in certain doubtful cases seems highly probable.

Dr. Russell tells me that he has already made 1,000 moving pictures of the interior of the normal larynx, largely in singers. Thus is being opened up a large new field of study. I think that we are to be congratulated for having the privilege of being present at an important historical occasion in laryngology.

DR. ROBERT SONNENSCHN: There is very little I can add in the way of admiration for the marvelous work Dr. Russell has done. It seems to me that we have in these pictures a proof of the fallacy of the former assumption that in the movements of the larynx during swallowing the epiglottis folded backward and closed the glottis. These pictures do not show the act of swallowing, but I take it that in some of the movements of speech the motion is almost the same as in swallowing. For a time it was

thought that the movements of the arytenoids helped to close the larynx during swallowing. Then again, a few years ago, Dr. Mosher presented a very fine paper in which, from X-ray studies, he assumed that the epiglottis covered the larynx in the act of swallowing. I think in these pictures we see that it is the movement of the arytenoids and the false cords that serve this purpose.

DR. ALFRED LEWY: I do not see how these pictures can show one thing very well, and that is the up and down movements of the larynx which occur during phonation. I do not know how Dr. Russell can show that, in addition to the anteroposterior and lateral movements, but perhaps he can devise something that will also show those movements.

Also, in the use of the laryngeal mirror we do not find that in most cases the attempt to phonate the vowel "e" gives us the best view of the interior of the larynx, but of course conditions are changed by our traction on the tongue. Sometimes the "hi" and sometimes the "e" is better. That is another demonstration of the fact that by using the tongue traction as we have to we are disturbing the normal relation of the larynx.

I wish to take issue with Dr. Kenyon on the fact that I have always felt that the patient was always conscious to a certain extent of the movements of the larynx by something like kinetic sense; that we are not dependent entirely on audition. That has been told me repeatedly by singers, who have said their vocalization "did not feel right." This has impressed upon me that there is some sensation akin to kinetic sense in addition to the auditory sense.

DR. ELMER L. KENYON: I agree with Dr. Lewy. There is no question, however, but that the statement I made is essentially true. We get these movements in the larynx primarily by thinking of the sound. However, there is a distinct sensation related especially to the mouth mould required for the sound, and, as I believe, to the action chiefly, not of the vocal cords themselves or of their muscles, but related to the extrinsic muscular movements. Undoubtedly such sensations are of great value to singers, and they also serve primarily to enable the person to acquire speech.

DR. J. HOLINGER: Years ago I was in a position to amputate the epiglottis on account of tuberculosis, often down to the lowest ridge and no loss of function could be noticed. The swallowing was often greatly relieved and no food entered the larynx. I came to the conclusion that we overestimated the importance of the epiglottis. All of these cases now go to the tuberculosis institutions as soon as we report them, and we do not hear any more of them. A large material must accumulate there. So far in our society I have not heard of any scientific contribution on the subject that would be adequate.

DR. G. OSCAR RUSSELL (closing): The pictures we have shown tonight form a part of a larger study financed by the Carnegie Foundation and conducted in collaboration with members of the American Academy of Teachers of Singing and of prominent opera and other vocal stars of this country and abroad. The purpose was to establish what the physiologic causes of voice quality differences are. The major part of these experiments consisted of X-ray pictures taken at a high rate of speed. A large number of subjects were utilized, including Althouse, Bori, Gigli, Johnson, Tibbett and many others of recognized vocal ability. By our X-ray technic we have been able to devise a setup which makes it possible to compute the exact length of the vocal cords. Likewise to indicate the rise and fall of the larynx; also opening and closing of the velum, dimensions of the vocal cavities themselves and of the openings, including those of the lips. All of these may be measured accurately to a fractional part of a millimeter. It will be seen, therefore, that the best information bearing on the question asked would be obtainable from our X-ray studies rather than from the moving picture vocal cord studies presented tonight.

The only way we would have of ascertaining whether the larynx rises or falls by study of these motion pictures of the vocal cord (since they are from a superior laryngeal position) would be by observing the field; and also by studying the changes in focus. That would not be anywhere near as reliable as the X-ray pictures in question.

As to the point raised by one other question, I do not believe it is possible to find a subject who will be able to articulate a

clear vowel "ee" during the old style laryngoscopic examination in which the ordinary laryngeal mirror is inserted and the light is reflected in by means of a condensing head mirror, nor by some of the more recent laryngeal mirrors which attach a light at the distal end of the handle immediately in front of the laryngoscopic mirror. The reason is obvious when you recall the X-ray pictures included as a part of the demonstration film tonight. For the vowel "ee" requires a very small buccal opening, normally no larger than your finger. And since this curves upward, it is not possible to see through it into the back pharynx. When this question first arose I verified that observation by attending a large number of examinations by different doctors where the attempt was made to get the subject to articulate a clear "ee." I believe I could say that though the subject often thought that was the vowel he was producing, something approximating "uh" was invariably produced in lieu of the "ee." I think one reason, therefore, why I should stress this fact is to get your reaction as you go back and listen carefully for the sounds you hear and suggest that I would like to have that reaction, particularly as you may obtain it from your office assistants, nurses and strangers who are not expecting any particular sound. For the fact is that we usually hear what we are "set" to hear. I dwell at length on this question because tonight's demonstration film makes it very clear that a vowel "ee" pronounced with unmistakable quality is necessary if a complete view of the interior larynx is to be had.

I was much interested in Dr. Kenyon's note to the effect that he had assumed long ago from exterior palpation and other methods that the larynx changed position rapidly in passing from one vowel sound to another, because when I published the first still vocal cord pictures of unhindered vowel positions in my book, "The Vowels," I was taken severely to task by a few who insisted that the vocal cords did not look so long as they there appeared. The reason is obviously because of the fact that all those taken formerly were with the ordinary laryngoscopic mirrors, resulting, as I have just pointed out, in necessity for the subject to articulate a vowel "uh." As you noted, this vowel, "uh," invariably blocks off a substantial part of the vocal cord view. It is not possible to see the anterior commissure and the area well up under

the pulvinar. You will have observed in tonight's demonstration that these motion pictures verify the earlier still photographs. In all cases where a clear vowel "ee" is articulated, the full length cord is shown in its stretched out appearance, and the whole interior of the larynx as well as the pharynx is seen to change as we pass from vowel to vowel.

I am delighted to have the opportunity to show you the pieces of apparatus I have devised for examination of the larynx. I demonstrated them last year and am sorry there is no time to demonstrate tonight. If there were, I believe you would agree that the Fonofaryngoskop, manufactured by the Electro Surgical Instrument Company in Rochester, gives the sharpest and clearest view of the cords you have ever had. Personally, I prefer to use my little Nongag Glottoskop for ordinary laryngeal examinations of such subjects as most laryngologists would encounter. I think all will agree that a clear ordinary laryngoscopic view of the cords is often impossible by reason of the tendency of some of the subjects to gag. In the Speech Clinic I have yet to encounter, however, any subject in whom a clear view cannot be had with the Nongag Glottoskop. You will notice that its lens and lighting system are of such a nature that a brilliant image is had, even though the tube is not very much larger than a match. It likewise is manufactured by the above mentioned company. Just as the Holmes' Nasopharyngoscope was a modification of the earlier Hayes' model, so likewise this is a modification. If you attempted to use either of those, the focus would be bad for they have entirely different lens and lighting systems, so you would be unable to get any kind of an adequate view of the cords. The lens system is rather elaborate in this little piece of apparatus, and while the view of the cords is smaller than that obtained with the Fonofaryngoskop, the big advantage that it has in permitting an examination of all subjects, including gaggers who heretofore blocked our attempts, is one that I have felt to be recommendable, and I agree with Dr. Kenyon that it is the most valuable of all the laryngeal pieces of apparatus I have devised. You will notice that the subject may close his lips and can articulate practically any speech sound. Its usage with an "m" therefore makes it possible to also obtain a sharp and clear view of

the whole nasopharynx viewed from beneath, so that for adenoid examinations it likewise has very distinct advantages.

The Practical Value of the Audiometer.

G. HENRY MUNDT.

(This article published on Page 215 of this issue.)

DISCUSSION.

DR. LOIS D. GREEN: In collaboration with Dr. Shambaugh, I am attempting to clarify some of the facts of bone and air conduction as recorded by 1-A audiometer and thus give added practical value to the audiometer examination.

The customary audiometer curve I shall designate as the standard curve and it is taken with the head phones in contact with the head and cushioned with sponge rubber. This is the curve ordinarily designated as air conduction. In cases of otosclerosis or other conduction deafness, the picture given by this curve differs very markedly from that given by the whispered voice and tuning forks by air conduction. In order to give a truer picture of the hearing loss by air in these cases, I began taking audiograms holding the receiver as near to the auricle as possible, but without any contact with the head. These I designated pure air curves and they were made on normal subjects, patients with conduction deafness and those with nerve deafness. They were plotted on the same charts with standard curves (phones in contact), and bone conduction curves made by the Western Electric bone conduction receiver No. D 80904.

In the course of a year certain points have become apparent.

1. The curve for pure air in normal subjects is 20-30 sensation units lower than the standard curve for frequencies up to 1024 d. v. or, more usually 2048 d. v., when it becomes only about 5 units lower and parallels the rest of the standard curve closely.
2. In a true conductive lesion, the pure air curve is 40-60 units lower than the standard curve for the lower frequencies and again approaches to within 5-10 units of it at 2048 d. v. This proves a truer picture of the useful hearing of a patient with a conductive lesion.
3. In a true nerve lesion the pure air curve bears the same relation to the standard curve that it does in the normal ear.

4. When the bone conduction receiver is used the results are apparently not reliable beyond 512-1024 d. v. unless the patient is very deaf. At these points the instrument is emitting so much sound by air that the curve is almost the same whether the apparatus is in contact with the head or slightly removed.

5. In cases of distinct conduction deafness, the curve produced by the bone conduction receiver may rise above the standard and pure air curves and approach that of normal hearing. In cases of nerve deafness it bears the same relation to the other curves that it does in the normal, being 20-40 units below the standard curve at 32-64 d. v. and about 60 units below at 512-1024 d. v.

These findings suggest that the standard curve with the phones in contact with the head does not represent the true air conduction but that a true air conduction curve will have to be developed and standardized. They are also of aid in visualizing and recording more accurately various types of deafness. Possibly additional light will be thrown upon the Rinne and Schwabach tests, particularly if the work can be done in a sound proof room.

DR. G. OSCAR RUSSELL: I think Dr. Green's contribution is very vital and believe that much more work of this sort is needed. In one of the papers I presented before this society last year, I showed a hearing test graph which could advantageously be used in lieu of an audiogram, to which perhaps you will pardon my calling your attention again. It is used as is the latter with any Western Electric 2-A or 1-A audiometer, but has an advantage over the audiogram in that it shows at a glance the exact status of the subject's hearing in terms of hearing remaining and lost, for each tone and the whole or any part of the range so that the median or peak is also graphically portrayed without any computation or reference to a conversion table. These advantages over the audiogram need no comment. I believe that Dr. Bunch and Dr. Shambaugh, who have protested some rather loose usage of hearing loss computation on the audiometer, are justified. The former has made a particular point of the fact that the customary statement of hearing loss is not adequate. One principal reason why this is true is that the term represents a too vague generalization. This is particularly so when one follows the recommend-

ed procedure of choosing three tones in the middle of the spectrum and averaging these with an arbitrary correction for a statement of what hearing is remaining in the subject. We long since observed that pedagogically, at least, all kinds of false conclusions are thereupon drawn. It is admissible that this statement is infinitely superior to what was formerly available, but surely we should not be content with anything less than the best. Since it is inadequate, it would seem advisable to cease the usage of this statement of hearing loss. There may be times when a statement of the median loss through the whole audiometer range would suffice, and might stand as a statement of the individual's hearing status. But the question arises in that connection whether it would often not be better to use the peak loss as a criterion by which to judge. Certainly it is true that for pedagogical purposes it is often absolutely necessary for us to know whether the loss is in the low frequencies or high frequencies, and the teacher must of necessity know whether the loss in the low frequencies is serious. Over 40 per cent of the meaning of speech is carried in an intonation rise and fall which involves the area from 60 to 256 cycles in male voices, and this is precisely the area which is left out of consideration in the usual statement of hearing loss computed on the basis of the three tones above mentioned. On the other hand, if the teacher is engaged in correcting speech difficulties it is quite essential that she know the status of the individual's hearing over the range from 3000 to 8000 cycles, for this is precisely the range involved in the characteristic tones required for the production of such sounds as "s", other fricatives and even most unvoiced stops.

The doctor cannot be blamed for not desiring to waste the time necessary in order to refer back to a conversion table and compute the hearing loss for each individual tone. This is the principal reason why we prefer the hearing test graph above mentioned, in lieu of the audiogram. It shows everything the audiogram does, and at the same time indicates at a glance just exactly what the hearing loss is for any particular range. It can be used for either the 1-A or 2-A audiometer. The patient who takes such a graph with him is better directed, and this is particularly true of children whose teachers need to know what can and can-

not be done in the matter of training and retraining the sound patterns involved in a cerebral hearing loss, which must inevitably follow a functional loss if nothing is done. I believe otologists generally could render a great service to the people of this country if they would all give their full support to recent moves they have fostered which tend in that direction. I refer particularly to those sponsored by Dr. Hayden and others behind the work of the League for the Hard of Hearing.

I should not close without calling attention to the phonograph speech tests now available through the work of Dr. McFarland, since through their usage considerable information may well be added to our present stock of knowledge in regard to the cerebral deafness.

DR. G. W. BOOT: I remember some years ago hearing a highly scientific paper read before the Chicago Ophthalmological Society on the proper method of illuminating a test card. The essayist proved by calculus and demonstrated by means of a graph that the only correct method of illumination was by means of a parabolic mirror.

In this highly scientific age if an article is not illustrated by one or more graphs it is discredited, and if the author of a paper furnishes the requisite number of graphs it is accepted as scientific.

I often wonder how Darwin managed to put his great work across without a graph to illustrate it.

The audiometer has two of the modern requisites; one always has a graph to show and one uses that modern method devised by Henry Ford and introduced into medicine by the famous clinic that habitually introduces its papers by the statement "in our last 10,000 cases," or words of similar import—mass production.

DR. ROBERT SONNENSCHN: I always hesitate to say anything about the audiometer because the impression has gone abroad that I am prejudiced against it, but this is absolutely not so. I think the audiometer has certain advantages, and feel that Dr. Mundt is to be congratulated for his fair way in presenting it. Most persons say we cannot practice otology without it, forgetting that innumerable individuals were examined successfully before it was devised. In many ways it is practical from the

standpoint of speed, ease of manipulation, etc., as compared with other methods in vogue.

First, let me call your attention to the fact that while the audiometer is an instrument of precision, and I have always been in favor of such devices and methods, the various types of audiometer are not calibrated alike. The Western Electric is best known, and most widely used. It is calibrated so that it tells the loss of hearing in sensation units, and to get percentage of hearing loss you multiply the number obtained by 0.8. But the calibration of the Jones-Knudsen, of the Krantz, the Dean-Bunch, and the Gutman audiometers (the latter it seems was the first one to use the vacuum oscillator tube in connection with the audiometer), are entirely different. I do not know about the physics of the subject, but am told that the curves or graphs of the different apparatuses are not comparable. It is true that it is nice to have graphs to show the differences in hearing at various times, but it is well not to forget that no less an authority than Harvey Fletcher stated that if we have the so-called "constant" of damping we get the same data regarding loss of sensation units with tuning forks that we do with the audiometer. The calculation is very simple and he gives the formula. So if you have four, five or six calibrated forks you can get the same curve as with an audiometer. While I have always advocated that we should excite forks uniformly, it has been shown that the decrement or rate of damping of the fork is always the same even if there is some difference in the force used. The formula is, loss of sensation units = $\Delta (t - t')$. Δ is the constant of damping; t = normal hearing time; t' = hearing time of patient. Let us say the decrement for the small a-1 is 1.5; the time the patient should hear it is 100 seconds; if you find the hearing is only 70 seconds you simply subtract this from the 100 and the difference is 30; multiplying this by 1.5 gives 45 sensation units lost. If you have the forks you can get the same graph that you do with the audiometer, but if you use many forks it takes a little longer. You can get the same graph you get with the Western Electric, for it is calibrated by the same system, so there is no reason for saying we cannot get the same graph with the forks as with the audiometer.

I must take a little exception to Dr. Mundt's statement that the Bezold forks are the only ones with which tests can properly be made, for I am sure you are aware of the work of our committee of the American Academy of Ophthalmology and Otolaryngology, and know that the forks prepared by Mr. Eisenbaur of the Riverbank Laboratory give the same data at a much lower price. I do not say this to deprecate the Bezold forks, but you can get the same results with new magnesium alloy forks which are rust and tarnish proof, and weigh only one-third as much as steel.

Dr. Mundt quoted from Dr. Shambaugh's very excellent paper which was read in Copenhagen. He stated that for the lower tones the audiometer is of no particular value; for the middle tones it is perhaps superior to the forks, and for the highest tones he thinks it is better than the forks. Recently Mr. Eisenbaur devised sounding rods of a pitch of C^3 (4096 d. v.) which vibrate from fifty to seventy-five seconds, whereas the best tuning fork of that pitch rarely vibrates longer than thirty seconds. He also perfected a rod of the pitch of C^6 (8192 d. v.). It is quite true that the earliest evidence of a change in the nerve of hearing is in the appreciation of the high tones, but the exact amount of hearing does not make any essential difference; all you need is an approximate idea. If the loss of hearing is very marked you can tell it in the course of ordinary conversation. By using the residual air in testing with unaccentuated whisper or conversation, you get a very practical estimate of the patient's hearing. Dr. Shambaugh also has pointed out this fact, that you do not need any apparatus for this purpose. The audiometer is a good apparatus; it is fine for the study of the physiology of hearing and for research, as Dr. Shambaugh has shown, but I still contend that with a few good forks, and tests properly made, we can get a good practical idea of the degree of hearing ability. Dr. Green demonstrated her work to me this afternoon, and while telephone men tell us that we get only air conduction with the receiver against the ear that is not entirely true, for we get some bone conduction as well as air. We know that if we hold the receiver against the ear we hear better than when it is out of contact with the auricle.

DR. G. HENRY MUNDT (closing): I think this type of discussion should continue; it is very wholesome. I do not stand before this organization as an advocate of the use of the audiometer.

Taking up Dr. Green's discussion, the standard graph undoubtedly is a very valuable thing. That will be worked out and I have no doubt many of us will have audiometers which are far superior to this 2-A audiometer. Such work as Dr. Green is doing in an effort to get pure air conduction is very valuable. From a purely practical standpoint with one patient I believe that the graph made today and made again in a few weeks is very valuable. Sound proof rooms are not easy to produce in Chicago. I have tried it and think Dr. Green will have some little difficulty, unless she gets some distance from the ordinary transportation units. There is no question that hearing tests of any kind should be made in as nearly a sound proof room as possible. I am perfectly willing to admit that when one figures the loss of hearing on an audiogram he has not a definite method. That hearing loss is, I think, much better measured in the average individual by the voice test, but not the evaluation of progress in a patient, here the audiometer is superior.

I appreciate Dr. Sonnenschein's discussion, and he will find, if he reads my paper, that I said that as a minimum type of fork we should have the Bezold. I had in mind, and I think I brought it out as to be easily understandable, that as a minimum type of fork we should have the forks of the Bezold type. I was trying to eliminate the use of two or three small forks, which are of little value. To be more specific, the set commonly sold as Hartmann's tuning forks. I said in this paper, and have always maintained, that in determining the loss of hearing of an individual the voice test is probably more valuable than the audiometer, and yet for comparison, from time to time, the audiometer is the most valuable.

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